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WILLIAM J. GIES TESTIMONIAL DINNER

To attempt to inform the dental profession at large of the activities of William J. Gies would be presumption. His labors have resulted in broad changes in dental thought, dental education, dental journalism and dental science as a whole. It is these accomplishments that tell the history of his sacrifices for his adopted profession. We often hear Dr. Gies spoken of as a layman who has labored for dentistry. While Dr. Gies does not hold any university dental degree he has become a dentist in a far broader sense; he has taken upon himself to be a crusader for the highest ideals in our profession.

The American College of Dentists, American Dental Association, Canadian Dental Association, American Association of Dental Editors, American Association of Dental Schools, Dental Section of the American Association for the Advancement of Science, *International Association for Dental Research*, National Association of Dental Examiners and Omicron Kappa Upsilon are tendering a testimonial dinner to Dr. William John Gies in Atlantic City, July 11, 1937. It is planned at that time to announce the establishment of an Endowment Fund for the JOURNAL OF DENTAL RESEARCH as a perpetual tribute to William J. Gies. While we cannot hope to honor Dr. Gies beyond those attainments that reflect his name and deeds, we know that every member of the *International Association for Dental Research* and every reader of the JOURNAL OF DENTAL RESEARCH will make a sincere effort to attend this dinner and pay homage to the man whose idealism has brought greater realization of the significance of dentistry.

H. B. G. R.

A COMPARISON OF CRUDE AND REFINED SUGAR AND CEREALS IN THEIR ABILITY TO PRODUCE IN VITRO DECALCIFICATION OF TEETH[1, 2]

T. W. B. OSBORN, J. N. NORISKIN AND J. STAZ

From the Department of Physiology, University of the Witwatersrand, Johannesburg, S. Africa

In another paper (1) which deals with the correlation between the diet and dental caries in the South African Bantu the proposition is advanced that refined sugar and/or refined flour cause dental caries, but that these carbohydrates, as they occur in nature, are accompanied by a "protective agent" which is removed, to a greater or lesser degree, in the course of refining—that, in effect, these carbohydrates are a danger to the teeth, that nature makes them up with an antidote, and that refining removes this antidote. A series of *in vitro* experiments were carried out to test the validity of the propositions.

The authors were faced with the ubiquitous problem of how far *in vitro* results may be trusted as an index of *in vivo* processes. In the case of teeth they were more hopeful of their reliability than they would have been in the case of other organs. The fact that the enamel is laid down before the eruption of the tooth, gives added confidence in *in vitro* experiments. It is probable that there are several reasons for dental decay. It is to be expected that a severe disorganisation of the calcium and phosphorus metabolism of the individual would cause decay and that other forms of malnutrition may leave the teeth an easy victim to the forces of attack. The work of Mellanby and her collaborators has shown the importance of vitamin D in the development of teeth. It is equally certain that, even where there is no deeply hidden constitutional sabotage at work, teeth decay because

[1] Presented at the 15th Annual Meeting of the International Association for Dental Research, Baltimore, March 13–14, 1937.

[2] Most of the expense of this work was defrayed by a grant from the Research Grant Board.

they are assailed on the frontiers they expose to the food in the mouth. Both the following propositions may be put with full justification: a. that some teeth decay because they were poorly made or because they are weakened from within, b. that some teeth decay because their exposed surfaces are attacked by acid-producing bacteria. They are complementary and not contradictory. It is probable that some teeth are so weak that the most innocuous food debris will rot them; it is probable that the strongest teeth will succumb to some surface attacks; and it is probable that the two factors often work together. The authors, in concerning themselves mainly with the second proposition, do not wish to deny the validity of the first. Being concerned principally with events occurring on the surface of the tooth, they feel they have further reason to expect events *in vivo* to follow the line indicated by *in vitro* experiments.

In spite of this the authors emphatically do not advance dogmatic theories on the basis of *in vitro* results. Any speculative or tentative propositions may be advanced require the fullest possible *in vivo* testing before they can be accepted as anything more than theories.

EXPERIMENTAL

Teeth were induced to decalcify by incubation in mixtures of sugar and saliva or cereals and saliva. In the early experiments the teeth were cut into discs so that parts of the same tooth could be put into the control and experimental mixtures. The cut surfaces were sealed with cement, only the surface normally exposed in the mouth being in contact with the solution. The media were changed daily and were agitated by a stream of air bubbles. An attempt was made to discover the decalcification by measuring the tooth and by X-rays. These refinements of technique were discarded as they were found to give no advantage over the simpler technique subsequently adopted.

Undecayed extracted teeth were immersed in media obtained by chewing or rinsing the mouth with: a. normal saline, b. crude sugar-cane juice,[3] c. a 12 percent solution of refined white sugar, d. a cooked 15 percent whole cereal "porridge" (the whole meal was made by grinding the cereal grain in a coffee mill), e. a cooked 15 percent "porridge" made from best white flour or mealie meal. The strength of 15 percent was chosen for the cereals as this gave a "porridge" of a normal consistency. A pinch of common salt was added to the "porridge" because there is some idea that sodium chloride assists the amylolytic action of the ptyalin.

In each experiment the subject rinsed his mouth thoroughly with water, took a comfortable mouthful of the solution or porridge, chewed it or rinsed with it for two minutes

[3] Obtained through the courtesy of the research laboratory of the S. A. Sugar Association and containing about 12 percent sucrose. Since this is a highly fermentable material it was boiled immediately after being drawn off and was subsequently kept in a refrigerator.

and spat out the mixture. He then washed his mouth out again, waited ten minutes and repeated the performance with the next solution or porridge. In case the sequence should have an effect, in alternate experiments the solutions or porridges were taken in the reverse order (e.g. in one experiment they chewed in the order saline, whole wheat porridge, fine flour porridge, in the next they were taken in the reverse order).

The teeth used were undecayed, normal teeth and were obtained from patients who had been advised to have multiple extractions. Special attention was paid to the distribution of the teeth in the different media in each experiment. As far as was possible each tube contained the same number of each sort of tooth i.e. incisors, canines, premolars and molars.

Each tooth was examined independently by two of the authors (J. N. N. and J. S.) before the experiment commenced and a record was made of its main features. After incubation at 37°C. for periods of from two to eight weeks it was examined again by the same observers, independently. To eliminate the possibility of bias the observers were given the teeth at random and they were given no information as to what media the teeth had been in. Using a sharp probe they looked for softening of the enamel and, by holding the tooth in a good light, they were easily able to see the white patches characteristic of decalcification when present. On probing, these patches were found to be friable. In the later experiments a groove was ground in the enamel, because it was found decalcification took place sooner and could be observed more easily on a worn surface. In some cases interim examinations were made during the course of the experiments. Finally the teeth were stained with Grenacher borax carmine. The decalcified areas were dull and stained, the healthy enamel remained glossy and unstained.

The observers reported, in the case of each individual tooth, either that: a. there was no decalcification, (−) b. there was a doubtful change, (±) or c. there was a definite change, (+). In most cases their findings agreed; where they differed the following system was used:—one + the other ± counted as change, one ± the other − counted as no change, one + the other — counted as no change. It was decided, for the purposes of the present work to divide the teeth into two classes only, the decalcified and the unchanged (the doubtful cases, as indicated above, being put into the second class). An attempt to record the degree of decalcification was abandoned.

The authors do not imagine, that they are dealing with *in vitro* caries. They go no further that to expect that that which produces decalcification in an extracted tooth is a likely caries-producer *in vivo*, and that conditions which fail to produce decalcification *in vitro* are not likely to cause decay in the mouth.

RESULTS AND DISCUSSION

The results obtained are recorded in Tables I, II and III. In the case of sugar they lend strong support to the proposition that refined sugar causes decay but that sugar, as it occurs in the cane, is nearly harmless.

The similar proposition that had suggested itself for the cereals—"that the process of refining removes some protective agency"—is

supported by Tables II and III. The naturally occurring carbohydrates of sugar-cane, of the mealie and, more especially of wheat, do produce decalcification. Therein lies a problem for some future year. The significant question which arises out of this work is why do the refined products produce the decalcification more rapidly than the crude. An obvious suggestion was that the refined developed more

TABLE I

Crude cane juice vs. sucrose solution

EXP. NO.	3	4	5	10	11	28	29	TOTAL
Saline and saliva:								
No. teeth	4	2	3	4	4	—	—	17
No. decalcified	0	0	0	0	0	—	—	0
Crude juice and saliva:								
No. teeth	4	3	3	5	5	6	6	32
No. decalcified	0	1	2	0	0	0	0	3
Sucrose and saliva:								
No. teeth	3	3	4	5	5	6	6	32
No. decalcified	3	3	2	3	3	0	1	15

TABLE II

Whole wheat meal vs. flour (60–70% extraction)

EXP. NO.	7	8	9	21	22	23	24	TOTAL
Saline and saliva:								
No. teeth	4	4	4	—	—	—	—	12
No. decalcified	0	0	0	—	—	—	—	0
Whole wheat meal and saliva:								
No. teeth	4	4	4	3	4	2	3	24
No. decalcified	4	3	4	1	0	2	0	14
Flour and saliva:								
No. teeth	4	4	4	5	4	3	4	28
No. decalcified	4	4	3	2	3	3	4	23

acid than the crude, but this soon had to be discarded since, in numerous pH determinations in the course of the experiments, it was found that all the mixtures reached an acidity of the order of pH 4–5 within twenty-four hours of the commencement of the incubation and remained at this point for the duration of the experiment. It therefore becomes incumbent upon the authors to explain different rates of decalcification in the same order of hydrogen ion concentration. As

a tentative hypothesis it is postulated that naturally occurring carbohydrate is accompanied, in sugar-cane and in the cereals, by a protective factor which exerts its effect otherwise than by controlling pH. They are not prepared to speculate as to the identity of this "protective agent."

The cane-sugar used in the experiments is of the order of 99.95% pure. The flour and mealie meal were the most refined obtainable,

TABLE III

Whole mealie meal vs. highly refined mealie meal (60–70% extraction)

EXP. NO.	2	19	20	25	26	27	TOTAL
Saline and saliva:							
No. teeth	4	—	—	—	—	6	10
No. decalcified	0	—	—	—	—	0	0
Whole mealie meal and saliva:							
No. teeth	4	3	5	4	4	6	26
No. decalcified	3	1	0	0	0	0	4
Refined mealie meal and saliva:							
No. teeth	4	3	5	3	3	5	23
No. decalcified	3	2	4	0	1	2	12

TABLE IV

Whole mealie meal vs. 90 per cent extraction mealie meal

EXP. NO.	41	42	43	44	45	TOTAL
Whole mealie meal and saliva:						
No. teeth	8	10	6	6	9	39
No. decalcified	0	1	3	3	0	7
90% extraction mealie meal and saliva:						
No. teeth	8	10	6	6	10	40
No. decalcified	0	2	3	4	0	9

in each case between thirty and forty percent of the grain was eliminated in the process of milling. Another series of experiments was performed to discover the properties of less highly refined mealie meal, a meal from which only about ten percent of the grain had been eliminated. The results of these are shown in Table IV and they indicate that this degree of refinement does not significantly increase the decalcifying potency of the mealie.

It must be stressed that the data reported in Tables I, II, III, and IV

do not permit of a comparison of the relative decalcifying potency of sugar, flour and refined mealie meal. There is great variation between salivas from different persons and between the resistence of teeth from various cases. In addition, there has been no standard time for the incubation in the different experiments; investigation is still in progress and experiments have been terminated in an arbitrary manner to test the effects of shorter and longer exposures. It is clear, therefore that comparisons can only be made between different tubes of the same experiment, i.e. between tubes containing saliva from the same donor, teeth from the same subjects and incubated for the same period. To make a comparison between refined sugar, flour and mealie meal a

TABLE V

Sugar vs. flour vs. refined mealie meal

EXP. NO.	46	47	48	49	50	51	52	53	54	TOTAL
Sugar and saliva:										
No. teeth	3	2	2	3	4	3	3	2	3	25
No. decalcified	0	2	1	0	1	0	1	0	3	8
Flour and saliva:										
No. teeth	3	2	3	3	4	4	4	2	3	27
No. decalcified	2	2	1	2	1	3	1	2	2	16
Highly refined mealie meal and saliva:										
No. teeth	3	3	3	3	4	3	3	2	3	27
No. decalcified	0	2	0	1	2	1	2	2	3	13

separate series of experiments was set up the results of which are reported in Table V. It appears that flour and highly refined mealie meal, are rather more potent as decalcifiers than sugar under conditions obtaining in our experiments.

The authors are now attempting to obtain some information as to the nature of the protective substance which they postulate.

SUMMARY

1. By incubation in media containing sugar or cereals mixed with saliva for periods of, from two to eight weeks, healthy teeth were induced to decalcify. 2. Refined white sugar produced this change in a high percentage of teeth, crude cane juice caused very few to

decalcify. 3. This strengthens the proposition that refined sugar causes caries but that sugar, eaten as it occurs in nature, does not. The suggestion that there is a "protective agent" in crude sugar-cane juice, which is removed in the course of refining, is advanced with the added assurance. This agent does not depend on an ability to prevent or reduce acid fermentation. 4. Whole mealie meal causes very few teeth to decalcify, highly refined mealie meal (60–70% extraction) produces this change in a high percentage of teeth. There is no significant difference between 90% extraction mealie meal and whole mealie meal. 5. Whereas whole wheat caused a high percentage of teeth to decalcify, highly refined white flour (60–70% extraction) attacked a significantly greater proportion of teeth. 6. By analogy with the proposition put forward for crude cane juice, it is suggested that the carbohydrate in those cereals occurs in nature with a similar "protective agent," which tends to inhibit its injurious action on the teeth. The fact that this is no more than a partial protection on the case of wheat does not affect the theory. 7. Of the highly refined substances dealt with, flour and mealie meal appear to be more potent in producing *in vitro* decalcification than sugar.

The authors wish to tender their thanks to Professors Evelyn Sprawson, J. C. Middleton Shaw and Dighton Stammers for their advice and encouragement; to Dr. Monk and the staff of the Dental School of the University of the Witwatersrand, and to the leading anaesthetists of this town and their nurses for providing them with extracted teeth; to Mr. H. H. Dodds of the S. A. Sugar Association Research Station for the supply of crude cane-juice; to Mr. Cyril Everett of Messrs. Macdonald Formam & Co., Ltd., for supplies of cereals; to the subjects who supplied the saliva, and to Mr. James Morris, for his painstaking assistance at every stage of the work.

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CALCIUM CONTENT OF STIMULATED AND UNSTIMULATED SALIVA

SIGNE JONSGAR

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The divergent figures in the literature for the calcium content of saliva made it desirable to determine the effects of different methods of collection of the saliva. Becks (1), Clark (2), and Hawkins (3) analyzed samples of "unstimulated" or "resting" saliva taken at least one hour after a meal and without undue movements of cheeks and jaws. Horton, Marrack, and Price (4) used acid drops to obtain saliva, while Karshan, Krasnow, and Krejci (5) analyzed saliva stimulated by the chewing of paraffin. McClelland (6) and Morris and Jersey (7) found that saliva was changed quantitatively and qualitatively by stimulation with paraffin, but calcium is not mentioned. The pH is increased during paraffin stimulation, as shown by Starr (8), Peabody, Hall and Lewis (9), Forbes (10), and others.

One hour after breakfast resting saliva was expectorated into a graduated cylinder. Immediately afterward a number of equal samples were collected during stimulation with different substances. 30 minutes later another sample of resting saliva was taken. The time of collection for each sample was noted. 5 c.c. samples of saliva were transferred into a Kjeldahl flask; about 50 c.c. of concentrated nitric acid were added, and the mixture brought to boiling. When half the volume was evaporated a further quantity of nitric acid was added to cool the mixture, and 0.5 c.c. of perchloric acid added. The liquor was then evaporated to about 1 c.c. by boiling. If the solution was still brown, more nitric acid was added; otherwise evaporation was continued until a white residue appeared and about 0.5 c.c. of the liquor remained. The solution was pipetted into a centrifuge tube, washing twice with 10 drops of distilled water, and the calcium determined by the method of Kramer and Tisdall (11).

In some of the cases saliva was not ashed, but centrifuged at 3500 revolutions per minute for 30 minutes. The saliva from children, clearer than that from adults, was centrifuged for 15 minutes only. The slightly opaque

TABLE I

Calcium content of saliva determined by direct precipitation without ashing. Each sample was 3 c.c. of which 2 c.c. were used for analysis. Subject S. J.

SAMPLE NO.	TIME OF COLLECTION (MINUTES)	CALCIUM (MGM./100 C.C.)		STIMULATED WITH
		Unstimulated	Stimulated	
1	20	6.0		.....
2			4.6	paraffin
3			3.9	"
4			3.9	"
5			3.8	"
6	7		4.1	"
7	4		3.7	"
8	3		4.0	"
9	7		4.3	"
10	7		4.6	"
11	25	6.3		.....
1	15	6.4		.....
2	4		4.3	paraffin
3	3		3.3	"
4	8		3.7	"
5	7		lost	"
6	8		4.2	"
7	8		4.3	"
8	9		4.2	"
9	7		4.3	"
10	3		3.7	"
11	40	6.3		.....
1	18	5.5		.....
2	0.5		4.7	sugar*
3	0.5		4.4	"
4	0.5		4.6	"
5	20	5.0		.....
1**	15	5.8		.....
2**	5		5.4	sand
3**	5		5.2	"
4**	5		5.2	"
5**	15	6.1		.....

* Crystallized pure sugar, free from calcium, was used.

** These samples were shaken with one drop of 5 per cent acetic acid before centrifugalization.

supernatant liquor was carefully poured off, and calcium precipitated directly.

The data in Tables I and II show that the saliva is changed quantitatively with respect to calcium with any type of stimulation. The

TABLE II

Calcium content of saliva determined after ashing. 5 c.c. samples were used for analysis

SUBJECT	SAMPLE NO.	TIME OF COLLECTION (MINUTES)	QUANTITY COLLECTED (C.C.)	CALCIUM (MGM./100 C.C.)		STIMULATED WITH
				Unstimulated	Stimulated	
S. J.	1	30	about 5	6.9		
	2	2	5.5		5.9	paraffin
	3	2	5.5		5.4	paraffin
	4	30	5.5	6.0		
S. J.	1	60	6	5.9		
	2	1.5	7		4.8	sugar
	3	3	7		4.6	sugar
	4	50	5.5	5.8		
R. N.	1	35	7.5	6.9		
	2	6	7.5		5.3	sugar
	3	7	8		5.3	sugar
	4	29	5.5	6.8		
F.	1	25	about 7.5	5.3		
	2	1.5	7		15.8[1]	citric acid crystals
	3	1.5	7		18.4[1]	citric acid crystals
	4	20	7.5	5.8		
F.	1	18	7	5.5		
	2	3	7		6.0	10 drops acetic acid
	3	4	7		6.8	9 drops acetic acid
	4	15	8	5.7		

[1] These high values are probably due to calculus deposits or tooth substance dissolving.

secretion rate is considerably increased, and the calcium content is lowered. These results are in accord with those of Clark (3), and Krasnow (12). Becks (1) reported values 7.6 and 17.5 mgm. per cent, with a mean of 12.13. The figures of Entin (13) average 15.9 mgm. per cent. It is difficult to understand how these high values were

obtained. Hawkins (2) reported figures between 2 and 7 mgm. per cent; Clark (3), between 4.6 and 10.8; Peabody, Hall and Lewis (9) found 5.3 and 5.1 mgm. per cent.

Effect on Salivary Calcium of Ingesting a Bone Preparation: From May to July 1933, about 60 analyses of calcium in resting saliva were made at the Children's Department, Norwegian Dental School, Oslo. The technic employed was direct precipitation of calcium as oxalate in the centrifuged saliva, as described above. The figures ranged from 3.3 to 5.9 mgm. per cent. The anlyses were made both before and after the intake of *Kalfocitt*, a special mineral prepared from bone. In

TABLE III

Calcium content of saliva before and after the intake of "Kalfocit" over a period of one month

SUBJECT	AGE (YRS.)	CALCIUM (MGM./100 C.C.)				
		Before "Kalfocit"	After "Kalfocit"	Difference (After-Before)	Milk intake per day	No. of cavities filled in 1933
A. K.	13	5.2	5.1	−0.1	1 pint	4
W. O.	14	4.2	4.8	+0.6	1 pint	0
M. H.	13	4.8	5.3	+0.5	½ pint	10
I. M.	13	4.1	4.7	+0.6	½ pint	10
I. O.	14	4.9	4.8	−0.1	1 quart	19
E. N.	6	5.0	5.7	+0.7	very little	15
I. S.	6	4.0	5.0	+1.0	1½ pint	12
A. S.	14	4.7	4.7	0.0	1½ pint	16
A. S.	14	5.1	5.4	+0.3	1 quart	16
Average.....		4.6	5.0	+0.4		

most of the cases the intake of this preparation over a period of one month increased the calcium in saliva. (Table III.)

The daily intake of milk by these children was noted. Where the milk consumption was 1 quart per day, supplemented with a teaspoonful per day of the bone preparation containing about 1 gm. calcium and phosphorus per day, the inorganic salts should be abundantly provided for. Still the figures do not exceed 5.9 mgm. per cent. The figures are the average of 4, 3, and 2 single analyses, samples being taken on different days. Resting saliva was used, and the samples were usually taken at 11 A.M. at least one hour after a meal.

Conclusions

When stimulated by any of the methods employed, the flow of saliva was considerably increased, and the calcium content was lowered. Calcium in "resting" saliva ranges between 3.3 and 6.9 mgm. per cent; in stimulated saliva, between 3.3 and 5.9 mgm. per cent (excluding acid stimultion).

The calcium content of saliva increased somewhat by the intake of one teaspoonful per day for one month of a special bone preparation.

It is a pleasure to acknowledge the help and advice of Magister Nordbö. My thanks are also due to Professor Langfeldt, of the Physiological Institute, for providing place and opportunity to carry out this investigation, and to Professor Toverud of the Dental School, for suggesting the administration of "Kalfositt," a bone preparation which has been proved to be assimilated in animals and in human beings by Toverud and Toverud (14).

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COMPARISON OF DIURNAL AND NOCTURNAL pH VALUES OF SALIVA[1]

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Although the pH of diurnal "resting" saliva has been studied by several investigators, no data are available regarding the pH of saliva during the night, more particularly during sleep. That the pH range of saliva during the night may vary from that during the day is suggested by certain pertinent factors, namely, that saliva stagnates during the night, that there is an increase in bacterial flora, that basal metabolism and respiratory levels are lower—any or all of which factors may influence salivary pH. In the belief that a more complete knowledge of the pH of saliva might be desirable, this study was undertaken. It is possible that the difficulty in obtaining subjects for pH studies during the night, rather than a lack of interest, has deterred others thus far from pursuing this problem. We were fortunate in enlisting the support of dental students and of patients in a hospital, to whom we are greatly indebted.

The salivas of all subjects were studied hourly over a 24 hour period, with few exceptions, to determine the normal pH range during the day for comparison with that during the night.

The subjects may be divided into three groups: *Group I.* Apparently healthy subjects (the authors of this paper) who remained awake and active during a 14 to 24 hour period and whose salivas were studied during that time. At least one-half of the pH values were obtained from hourly samples of saliva during the night. Although there were only two subjects in this group, five studies were made at different times. *Group II.* Apparently healthy dental students. The pH of twenty-five salivas were studied in this group, of which three were duplicates taken about 10 months apart. *Group III.* Patients in the medical ward of a hospital. There were eleven subjects in this group. Subjects in Groups II and III were requested to go about their normal routine, except to refrain from eating or smoking for at least 15 minutes before a pH determination. They were also informed that the saliva desired for testing was resting saliva and that only a few drops

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[2] The authors are indebted to several members of the Sigma Epsilon Delta fraternity (Penna.) and Alpha Omega fraternity (Temple), and to the dental staff of Mt. Sinai Hospital for their cooperation in these studies.

were necessary, so as to minimize their drawing fresh saliva into the mouth just before a test. During the day the subject reported hourly at the pH station and expectorated into a dappen dish. The saliva from non-ambulant patients was collected by an assistant.

TABLE I

Average pH values of saliva while subject was awake (diurnal), while subject was asleep (nocturnal), and total combined values

	NUMBER	DIURNAL AVERAGE pH	NOCTURNAL AVERAGE pH	TOTAL AVERAGE pH
Group I	1			6.63
	2			6.65
	3			6.63
	4			6.78
	5			6.66
Group II	1	6.38	6.17	6.30
	2	6.75	6.27	6.55
	3	6.64	6.45	6.54
	4	6.63	6.09	6.42
	5	6.56	6.30	6.45
	6	6.93	6.56	6.82
	7	6.74	6.29	6.56
	8	6.60	6.14	6.41
	9	6.54	6.32	6.64
	10	6.76	6.51	6.68
	11	6.82	6.14	6.52
	12	6.52	6.13	6.31
	13	6.88	6.42	6.69
	14	6.68	6.38	6.51
	15	6.65	6.25	6.51
	16	6.99	6.49	6.83
	17	6.64	6.10	6.43
	18	6.23	6.06	6.19
	19	6.83	6.43	6.73
	20	7.17	6.70	6.95
	21	6.87	6.50	6.79
	22	6.92	6.50	6.78
	23	6.63	6.22	6.50
	24	6.85	6.30	6.69
	25	7.11	6.66	6.96
Group III	1	6.02	6.65	6.10
	2	5.91	6.04	5.95
	3	6.21	5.45	5.86
	4	6.45	5.88	6.25
	5	6.25	6.10	6.18
	6	6.10	6.45	6.19
	7	6.28	6.33	6.30
	8	6.32	6.03	6.21
	9	6.63	6.31	6.51
	10	6.72	6.19	6.50
	11	6.70	6.56	6.64

The amount of saliva expectorated varied from a few minims to more than 0.5 cc. The saliva was immediately examined, the time elapsing between expectoration and pH determination varying from about 30 seconds to 2 minutes. During the night, sleep was

interfered with as little as possible. The subject was awakened only sufficiently to spit into the dappen dish. With many, spitting had become a conditioned reflex and practically no time was lost from sleep. Several of the subjects experienced some dryness of the mouth during the night, but although the collected specimen was scantier than during the day there was a sufficient supply for a test. The saliva was examined immediately. The time elapsing between collection of the specimen and determination of pH never exceeded 3 minutes.

The apparatus used in the study was of the potentiometric type having a saturated calomel cell and a Cullen quinhydrone electrode. Because of the narrow bore of the Cullen electrode, only 2 or 3 minims of solution are necessary for a test. Loss of carbon dioxide from the saliva is minimized because the set-up forms a closed system. Before each series of tests, the apparatus was checked with a buffer solution of known pH.

TABLE II

Average pH values of different groups

GROUP	HYDROGEN ION CONCENTRATION
Group I	6.7
Group II	
Diurnal	6.7
Nocturnal	6.3
Group III	
Diurnal	6.3
Nocturnal	6.1

RESULTS

In *Group I*, in which the subjects were awake and active during the night as well as during the daytime, there was no apparent difference between what would correspond to the "diurnal" and "nocturnal" pH values of saliva. The average pH of this group for the entire period of study was 6.7. In *Group II*, composed of healthy dental students who averaged about 8 hours sleep during the night, there was an appreciably difference between the nocturnal and the diurnal pH values. The average pH for the group during the night was 6.3 as against 6.7 during the day. In *Group III*, composed of ambulant and non-ambulant patients, the nocturnal pH was also lower than the diurnal pH, being 6.1 and 6.3 respectively. The average pH values of this group both for night and daytime are lower than those in Groups I and II. That this is due to lessened activity is quite possible. At any rate, even though the salivary pH was low while the subjects in Group III were awake, it was still lower when they were asleep. (See Tables I and II, and *fig. 1.*)

CONCLUSIONS

1. There was no apparent drop in pH of saliva when the subjects were awake during the night. 2. There was a drop in pH during sleep in 32 of the 36 salivas examined hourly over a twenty-four hour period. 3. Our data suggest that the pH range of nocturnal saliva, i.e. during sleep, is lower (is more acid) than the diurnal pH range.

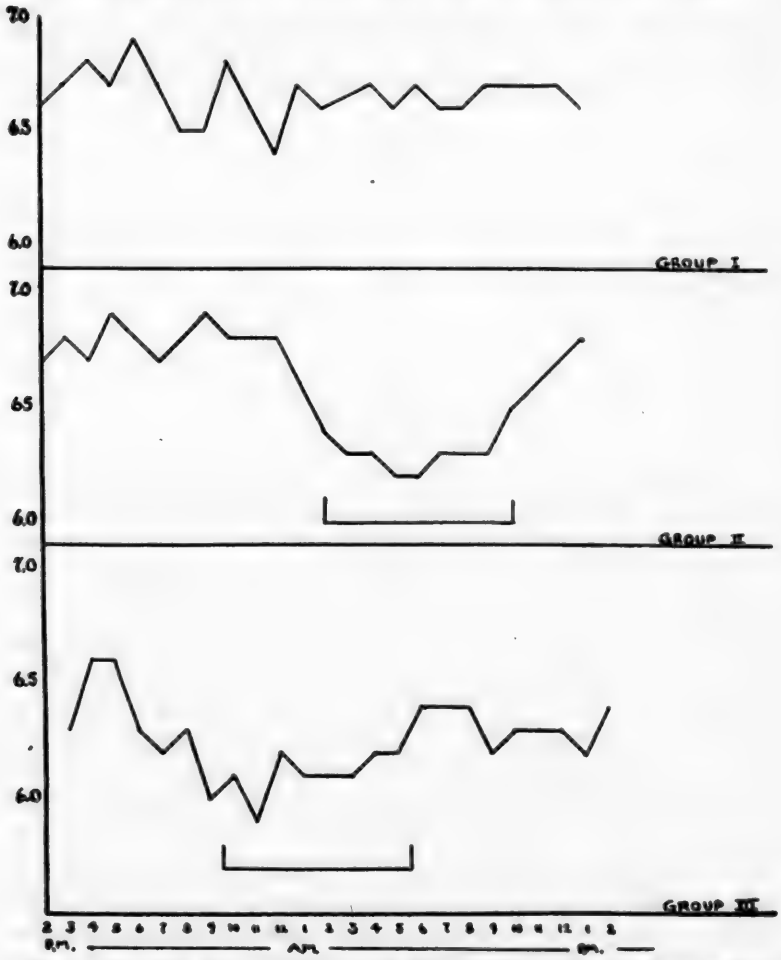

Fig. 1. pH values given at left, hour at which specimen was taken beneath chart. Brackets indicate time during which subject slept. All pH values average for group at time indicated.

A TECHNIQUE FOR THE PREPARATION OF HISTOLOGIC SECTIONS THROUGH TEETH AND JAWS FOR TEACHING AND RESEARCH

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The principal reason for the difficulties encountered in the preparation of sections through teeth and jaws is that the technique used is not adapted to the material. The fundamentals of the technique given in this paper were worked out in the histology laboratory of the Dental Institute of the University of Vienna under the direction of Prof. B. Gottlieb and, in 1925, were described in part by one of his technicians, Amalie Billig (1). Since then, especially in this country, modifications have been necessitated by differences in the stains, celloidin, and other materials obtainable here.[1]

Types of Material. This technique can be used for almost any kind of material, calcified or uncalcified, but it is especially adapted to calcified specimens, such as teeth and jaws.

The most easily obtained material is extracted human teeth, which are excellent for the study of normal dentin, cementum, and pulp as well as caries, secondary dentin, pulp atrophy, pulp stones, and various forms of pulpitis.

Animal jaws can be used for the study of the normal histology of the tooth, periodontal membrane, bone, and gingiva and for tooth development and shedding. Likewise, animals are invaluable for experimental work on diet and nutrition, endocrinology, orthodontic tooth movement, extraction of teeth, healing of wounds and fractures, etc.

Human jaws are naturally the best material on which to study both the normal and abnormal conditions that are found in human teeth and their surrounding structures. It is the only material which is satisfactory for the study of gingivitis, pyorrhea, granulomas, cysts, variations in the periodontal membrane, and any pathologic changes that occur in the supporting tissues of human teeth.

[1] We have been helped in working out these modifications by assistance and valuable and helpful suggestions from the laboratories of Dr. W. G. Skillen at the Dental School of Northwestern University and Dr. I. Schour at the Dental School of the University of Illinois.

The difficulties in securing human jaws are well known. Material taken from cadavers that have been fixed *in toto* for anatomic dissection is unfortunately worthless, for the tissues of such jaws are too poorly fixed or too shrunken for histologic study and do not stain well. On the other hand, material obtained during post mortem examinations, even though the autopsy was performed four or five days after death, is valuable. If the body was kept in a refrigerator, and if the jaws are put into formalin within a few hours after the autopsy, the histologic sections made from them are usually very good. The excellence of the stained sections depends far more upon the technique employed than upon the number of days that elapsed between death and tissue fixation.

If, however, whole jaws from autopsies cannot be procured, there are methods of obtaining pieces of jaws from living persons (2, 3, 4, 5).

Recording of Data. The more data that can be acquired about the specimens, the greater becomes the value of the collection of slides in any laboratory. All specimens are given a number, or mark of identification, as they reach the laboratory. Individual teeth or other tissues together with their number are tied into squares of gauze. Animal heads or human jaws are marked but not given numbers or put into gauze bags until they are cut into blocks. The number for each specimen is written with India ink on narrow adhesive tape, folded to avoid having the sticky surface adhere to the specimen or gauze. The number on the cloth is still legible even after a year or two in the celloidin. In a notebook, under the number of the specimen, are kept the history of the case, the patient's name, and date. Several samples from our protocol book, giving the specimen, its number, and the date when it was placed in the various solutions, are:

SPECIMEN	NO.	FIX.	ACID	Na_2SO_4	H_2O	50%	70%	95%	ABS.	ABS.	ABS. E.	6%	12%	25%
Jaw 30 Man, 64 yrs. $\underline{765}$|	2257	Fo. Alc	6–14	6–25	6–26	6–27	6–29	7–2	7–5	7–7	7–9	7–12	7–19	7–27
Single tooth	2534	"	8–20	8–28	8–29	8–30	8–31	9–3	9–4	9–5	9–6	9–8	9–14	9–20
Jaw 74 Man, 52 yrs. |$\overline{4}$	2772	"	6–3	6–18	6–19	6–20	6–22	6–24	6–26	6–28	6–30	7–3	7–11	7–19
Intact embedded |$\overline{8}$	2838	"	11–28	12–5	12–6	12–7	12–8	12–9	12–10	12–11	12–12	12–14	12–20	12–28
Adamantinoma	2867	"	—	—	—	—	—	1–20	1–21	1–22	1–23	1–25	1–28	2–2

Radiographs, photographs, and similar records are put in separate files.

Human jaw specimens are given special consideration, because they are usually studied many times, with a different problem in mind each time; at first, the thickness of the periodontal membrane under various occlusal conditions may be investigated; later, the pulpless teeth and granulomas may be studied, and still later in the same jaw the diseased gingival tissues surrounding the teeth may be examined. Therefore, as a matter of routine, all jaws are photographed, showing them in occlusion in front view and profile as well as occlusal views of both maxilla and mandible, and completely radiographed. Then, impressions of the jaws are taken with dentocoll and casts made, so that after the jaws have been decalcified, there can be no question as to their occlusion or original appearance.

The photographing, radiographing, and making of casts of the specimens are done after they have been fixed in formalin and while they are in 95 per cent alcohol. They are taken out of the alcohol only temporarily and never allowed to dry.

Fixation. For large specimens of dense, calcified tissues, the prime requisites of a fixing reagent are that it penetrate and fix all parts of the specimen as quickly as possible. Since formalin is powerful as well as rapid in its penetration, it is especially suited for the fixation of such material. A 10 per cent solution (250 cc. of 40% formaldehyde solution to 750 cc. distilled water) has consistently produced the best results. Other fixing reagents have been tried, but they seemed to have no special advantages for routine work. Since an intact tooth has only the small opening of the apical foramen through which the formalin can reach the pulp tissue, it is necessary either to drill a hole into the side of the tooth through the enamel and dentin or to cut off the apex, in order to have the fixing solution reach the pulp tissue before it begins to disintegrate. In teeth with large, open foramen or with deep decay this is not needed.

The jaws of animals, such as dog, cat, pig, sheep, monkey, or rat, are fixed immediately after death, simply by immersing them in 10 per cent formalin, or the fixing fluid can be injected into the whole head through the aorta or carotid artery. Human specimens, either whole jaws or blocks from jaws, are placed in a large jar of formalin as soon as possible after the autopsy or operation.

The amount of time required for adequate fixation depends upon both the size and density of the specimen; the examples given here may be used as a basis for comparison for other, similar material. Soft tissues, such as tumors, cysts, and small biopsies require only a day or two in formalin, depending upon the size. Individual teeth need from three to seven days' fixation: a child's tooth with large apical foramen or a deeply decayed tooth is more quickly fixed than an intact adult tooth with only a small hole drilled in the side for penetration of the fixing fluid. A week or ten days are adequate for blocks of one or two teeth with their surrounding tissues. Whole jaws, however, are left in the formalin for at least a month. It must be emphasized that no specimen can remain for months in formalin without the tissues becoming too hard and brittle, so that later they will neither cut nor stain well. Therefore, after having been fixed in formalin, the specimens are transferred into 95 per cent alcohol for about the same length of time as they were in formalin. At this stage of the procedure, or later in thick celloidin, the specimens may be kept indefinitely without harm.

Decalcification. After having been in 95 per cent alcohol for a sufficient length of time, the specimens are rinsed in running water for 24 hours. Five percent nitric acid is used for decalcification. Other acids and combinations of acids with fixing solutions have no special advantage over nitric acid and may even be destructive to the tissues. The teeth or whole jaws are suspended in a jar containing about five liters of the acid (75 cc. of concentrated nitric acid, Spec. Grav. 1.42, to 925 cc. of distilled water) and are left undisturbed. Agitation of the specimen, heating of the acid, and other methods of obtaining more rapid decalcifica-

tion have been tried but were found unsatisfactory. The best means of testing the specimens for complete decalcification is to pierce them with a sharp needle or steel pin: when no gritty substance can be felt, the decalcification is ample. Jaws are taken out of the acid after a week or ten days, cut into blocks of the desired size with razor blades (single-edged with a back), numbered, put into gauze bags, and then placed in the acid again until completely decalcified.

The amount of time required for the decalcification of several kinds of specimens when left undisturbed in a large jar of acid is as follows:

Single human tooth	7 to 9 days
Jaws of rat	24 hours
" " dog	7 to 13 days
" " new-born (human)	3 days
" " 2 months "	4 days
Jaws of 1 year (human)	5 days
" " 2 years "	6 to 7 days
" " 4½ years "	7 to 8 days
" " adults "	9 to 14 days

The time required for the decalcification of an adult human jaw varies according to age, sex, body size, etc. Small jaws from an older woman, for instance, may be completely decalcified in a little over a week, whereas the larger jaws from a man with heavy compact bone plates may require two weeks or even more. Decalcification also takes longer in acid in which several large specimens have already been decalcified, for the nitric acid becomes exhausted by use. From the nitric acid, the specimens are transferred into a 5 percent sodium sulphate solution (50 gm. sodium sulphate crystals to 1 liter distilled water) for 24 hours, and then into running water for 24 hours.

Dehydration. Dehydration is accomplished by carrying the specimens through rising percentages of alcohol—50, 70, 95 per cent and absolute alcohol. Single teeth or soft tissues are left one day in each percentage; pieces of jaws, animal or human, two days in each. Tissue that does not need decalcification is put directly from the fixative into 95 percent alcohol, and from then on is treated the same as other material. Two bottles of absolute alcohol are used to insure complete dehydration and anhydrous cupric sulphate covered with filter paper is kept at the bottom of these bottles to take up the water drawn from the specimens. Before embedding, the specimens are put in equal parts of absolute alcohol and ether for two days (three days for jaw material).

Embedding. Celloidin is excellent as an embedding material for teeth and jaws. Maximow (6) wrote: "The use of paraffin permits the tissues to be sectioned relatively rapidly and in very thin slices. Celloidin, on the other hand, disturbs the arrangement of the cells less and causes less shrinkage than does the paraffin method." Some technicians object to celloidin on the grounds that it is not possible to get as thin sections with it as with paraffin, but actually it is neither necessary nor especially advantageous to have sections so thin. A section of a

tooth embedded in celloidin and cut 12 micra thick in perfect condition is far preferable to one embedded in paraffin and cut 6 micra thick, but wrinkled and torn. Embedding with celloidin does take longer than any other method, but this is not so inconvenient as it sounds. If material is being run through the celloidin continuously, there is always some of it ready to be mounted.

Several kinds of celloidin are used in this country, each has its advantages. Celloidin shreds (Parlodion) are excellent, but for ordinary purposes their superiority is outweighed by their expense. Gun cotton has proved to be very good and is comparatively inexpensive.

The percentages of celloidin to be used vary according to the product, for instance, a 10 percent solution of Parlodion equals in thickness a 25 percent solution of gun cotton. Primarily only two facts need be known: one is that a thin, medium, and thick solution are necessary for proper embedding, and the other is that the longer the specimens can remain in celloidin, especially the thick solutions, the better. Small specimens are left in each celloidin three or four days, larger pieces, such as jaw material, at least a week in each. If it is possible, though, it is best to leave large, dense pieces in the thick solution for weeks or even months. Celloidin may be used many times and still give good results for routine material. When, however, it becomes yellow from the fats and oils of the specimens, it should be replaced.

The solutions of gun cotton[2] are made as follows:

25% (thick) solution
- —100 gm. gun cotton
- —103 cc. absolute alcohol
- —140 cc. ether

12% (medium) solution
- —100 cc. 25% solution of celloidin
- —100 cc. ether and absolute alcohol (equal parts)

6% (thin) solution
- —100 cc. 12% solution of celloidin
- —100 cc. ether and absolute alcohol

Mounting. Specimens may be mounted directly from 25 percent celloidin onto the fiber or wooden blocks. This method has disadvantages, though, for most teeth and large jaw sections. Usually a tooth has to be cut so as to get central sections through the pulp chamber and root canal, and since many teeth are curved or have spreading roots, this necessitates tipping the block to the correct angle and, if the tooth is lying directly on top of the block, the knife will cut the inclined block as well as the specimen. An indirect, longer process of mounting is required for such teeth and most jaw pieces. The specimens are taken from the thick celloidin, put top down in small glass dishes (such as Stender dishes) and well covered with new thick celloidin. The identifying number is put on the side of the dish beside each specimen; it is wet with celloidin and dries in place. The

[2] RS ½ sec. nitrocellulose, Viscosity 3–20—from Hercules Powder Company, Parkin, New Jersey.

dish is then covered and for three or four days is left undisturbed except for cutting around the edge with a knife on the second day in order to have the celloidin harden uniformly. When the celloidin is hard, that is, as hard as desired for sectioning, it is cut around each specimen, and the excess trimmed off with a razor blade. Thus, the specimen is oriented on a base of hardened celloidin. Before being placed on the block, the specimen is put in absolute alcohol-ether solution for a few seconds, or until the outer surface is slippery; some additional thick celloidin is necessary to affix it properly to the block. The blocks are put into chloroform for several hours, although air drying is often sufficient. Then, when the celloidin is sufficiently hard, the blocks are placed in either 70 or 95 per cent alcohol, where they may remain for weeks. If desired, glycerin in equal parts may be added to the 95 percent alcohol helping to keep the celloidin from loosening from the block.

Sectioning. A heavy sledge type of microtome is best for sectioning decalcified material (such as the Reichert OmE), and the longer the knife used the better. Large blocks of jaw material require at least a 185 mm. knife, but a 250 mm. one gives better results. The knife is adjusted to each specimen, so that the entire cutting edge is used. Also the angle of the knife is changed according to the hardness or density of the material to be cut: the knife is in an almost horizontal position for large decalcified pieces, at an acute angle for soft tissue, and halfway between for the average tooth. A slight variation in the angle will often determine whether or not the sections tear, as well as whether they will be thick or thin. During sectioning both the specimen and knife are kept wet with 70 percent alcohol. An overhanging drop bottle keeps the specimen from drying, an essential if the sections are to be of uniform thickness. Each section, as it is cut, is uncurled on the knife with a camel's hair brush, with which it is then put directly onto the slide. For routine staining, the sections are fastened on the slide with collodion. They are blotted with fine filter paper and then covered, but not flooded, with collodion. When the collodion wrinkles around the edges of the sections, the slide is put face down on the wet filter paper and pressed hard and evenly several times, after which it is put into distilled water for at least 24 hours before staining.

If the collodion used for this purpose is not the correct percentage, the sections will float off the slide when it is put into water. The following solution is used:

3 cc. Collodion, USP
27 cc. Ether and absolute alcohol (equal parts)

Removal of Celloidin for Special Stains. For special stains, such as Gram or azur-eosin, all of the celloidin is removed from the sections. A section is taken from the 70 percent alcohol in which it is kept after sectioning and placed on a slide that has a thin coating of egg albumin. Folded tissue paper is laid over the section and pressed down forcefully with another slide. Immediately after taking off the tissue paper, the slide is heated over a flame for a few seconds (as is done to fasten a smear to a slide). If is then put into 95 percent alcohol for a

few minutes, absolute alcohol for five or ten minutes, and absolute alcohol and ether for twenty minutes, which removes all the celloidin; then again into absolute, 95, and 70 percent alcohol for a few minutes each. It is kept in distilled water until stained.

Staining and Differentiation. Harris hematoxylin and acid-alcohol eosin are used for staining sections for teaching and for most of the research specimens. These stains are made as follows:

Harris hematoxylin

20 gm.	Potassium alum
200 cc.	Distilled water
1 gm.	Hematoxylin crystals
10 cc.	95 per cent alcohol
0.5 gm.	Mercuric oxide

By heating dissolve the potassium alum in the water. Add the hematoxylin crystals after they are dissolved in the 95 percent alcohol. Boil the solution quickly; then add the mercuric oxide (solution turns dark purple). Remove at once from the flame and put in a basin of cold water. Filter and put in stock bottle. For staining, dilute this solution using 20 cc. stock solution to 80 cc. distilled water.

Acid-alcohol eosin

2.5 gm.	Eosin (aqueous)
500 cc.	Distilled water
4 cc.	Concentrated hydrochloric acid

Dissolve the eosin in the distilled water and add the acid with a dropper. A precipitate will form and settle on the bottom. Pour off the water but do not lose the precipitate. Wash the eosin by filling the beaker at least six times, each time allowing the eosin to settle. Filter and dry the precipitate in a 52°C. oven for 24 hours. Remove it from filter paper and mix with 95 percent alcohol, using 0.5 gm. of eosin to 100 cc. of 95 percent alcohol.

The time required for staining with Harris hematoxylin varies greatly according to the age of the stain as well as the thickness of the section; usually 30 to 90 minutes are necessary. The appearance of the sections, using the microscope or not as one wishes, is the only means of judging when they are adequately stained. The slides are rinsed in tap water for 15 to 30 minutes, or until they are bright blue. After being placed in 50 and 70 percent alcohol for 10 minutes each, they are put for 2 or 3 minutes into the eosin, dipped for a second into acid alcohol (0.25 percent HCl in 80 percent alcohol), put into two 70 percent alcohols, and finally into 95 percent. Preferably the slides should remain in each of the last two alcohols for an hour; otherwise, what with the sections being comparatively thick and covered with collodion, they will not be sufficiently differentiated. The excess 95 percent

alcohol is allowed to drain from the slides before they are put between sheets of filter paper soaked with terpineol, where they are kept for one or two days. The slides are then put in a dish of xylene before the final step of dammar and cover slip. Dammar has proved to be preferable to balsam in that it dries more quickly and does not turn yellow with age. It is prepared by dissolving pure gum dammar in xylene until a syrupy consistency is obtained. If desired, the solution can be thinned by adding xylene or thickened by evaporation.

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INFLUENCE OF PARATHORMONE ON THE PRODUCTION OF DENTAL CARIES IN RATS[1]

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We have already reported data obtained from studies on the effects on dentition of alterations of calcium and phosphorus metabolism. (1, 2, 3). Here it was shown that disturbed inorganic salt metabolism is a very important factor through which the pulp and the integrity of the calcified tissues of the teeth may be affected. From the results obtained in our animal experiments, we have expressed our conviction that dental caries as a disease, manifesting itself clinically as a lesion of the enamel of the tooth, is usually associated with a distinct disturbance of the pulp (1, 2, 3). The latter was described as (a) pulpal engorgement; (b) serous degeneration of the odontoblasts; (c) fibrosis; (d) necrosis. In teeth displaying these pulpal pathologic changes, radiolucent zones were observed in the dentin and enamel, which in the radiograph, assume the characteristics of dental decay although, these teeth at first, present no gross surface lesions. It is only after a long continued pathology that lesions in the enamel can be seen clinically (3). We have found that by disturbing the calcium-phosphorus ratio in the blood, regardless of the mechanism, we could induce the described pulpal pathology, which on prolongation of the experiment is followed by enamel caries.

Since the calcium-phosphorus metabolism is greatly influenced by the endocrine glands, a study of the internal secretions seemed advisable. There has been much speculation as to the relationship of the endocrines to the erupted dentition. In 1911, Erdheim (4, 5) studied the effects of parathyroidectomy on the incisors of rats. He observed that the teeth became opaque and brittle; the enamel and dentin were hypoplastic; blood vessels were present in the predentin and bands

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were produced by variation in calcification, running parallel to the tooth surface. The histologic picture resembles the lesions we have found in rachitic animals (1, 3). Gies et al. (6) studied the effects of parathyroidectomy on the chemical composition of the teeth. They found that "removal of the parathyroids in such animals without material disturbance to the thyroids is followed by deficient calcification in practically every experiment." Considerable data have been accumulated on the action of large amounts of parathormone on blood calcium and skeletal structures. A careful search of the literature on the parathyroid glands did not disclose any experimental studies on the effects on the teeth of prolonged parathormone administration. Taylor and Weld (7) have studied the action of this hormone and emphasize the similarity in action between parathormone and viosterol in raising the serum calcium. Morgan and Garrison (8) have shown that the constituency of the diet is an important factor in the reaction of the animal to parathormone injection. Jaffe and Bodansky (9) in their study of bone lesions in experimental hyperparathyroidism state that: "Long continued injection (of parathyroid extract) leads to progressive decalcification and resorption of the existing bone, to fibrous replacement of the marrow, and to the production of the other features characteristic of osteitis fibrosa." The changes in the parathyroid glands in calcium disturbances have been studied extensively. Erdheim (10) reported hyperplasia of the parathyroids in rats suffering from rickets. Hyperplasia of the glands in human rickets has been described by Pappenheimer and Minor (11). Bischoff (12) asserted that hyperplasia of the parathyroids was indicative of hypofunction. A critical review of the subject including a discussion of the metabolism of calcium and phosphorus and the parathyroid glands has been published by Hunter (13).

In our earlier experiments we have observed a high caries incidence in young animals fed various rachitogenic diets and in older animals whenever their calcium-phosphorus metabolism was disturbed (1, 2, 3). Large amounts of viosterol were fed to several groups of rats in order to mobilize the calcium, already deposited, and thus bring about a decalcification of the teeth and osseous structures. All of these animals developed dental caries. In order to determine whether an excessive amount of viosterol is a specific causative factor in dental caries, or whether any agency which causes decalcification can produce these lesions, we conducted a series of experiments to study the effects of parathormone administration on the teeth of rats. Thirty-six rats, four weeks old, were divided into six groups containing six animals in each. Litter mates were evenly distributed.

Group I and II were fed diet A, which was high in calcium and low in phosphorus and deficient in vitamin D. Groups III and IV were fed diet B which was low in calcium and high in phosphorus and also lacked vitamin D. A normal diet (C), sufficient in inorganic salts and vitamins was fed Groups V and VI. In addition to the diets prescribed, each rat in Groups II, IV, VI received daily injections of parathormone. During the first fifteen weeks, each animal of these groups received subcutaneously 0.5 unit per 100 grams body weight. Then the parathormone administration was interrupted for two weeks, after which the dose was doubled and kept at the same level until the end. After two weeks of feeding, one animal from each group was sacrificed. The study was terminated at 150 days of experimental feeding.

Diet A (Groups I and II)		Diet B (Groups III and IV)	
yellow corn	75	yellow corn	75
wheat gluten	20	wheat gluten	20
calcium carbonate	3	potassium phosphate (basic)	3
sodium chloride	1	sodium chloride	1
ground alfalfa	1	ground alfalfa	1

Diet C (Groups V and VI)

yellow corn	73
wheat gluten	10
casein	10
calcium carbonate	2
sodium chloride	1
potassium phosphate (basic)	2
ground alfalfa	2
cod liver oil	3%

Several samples of ground corn, such as was used in preparing the diets, were tested for particle-size, the average of these being:

10 mesh	retained	0.7	per cent
20 "	"	12.0 "	"
30 "	"	18.0 "	"
60 "	"	28.0 "	"

In Group I which included animals fed a diet high in calcium, low in phosphorus, and deficient in vitamin D, and receiving no parathormone, the histologic findings were similar to those we have observed in other rachitic animals (2). The animal killed at the end of two weeks showed distinct pathologic changes as compared to the normal control of the same age. An incipient engorgement of the pulp was observed. The predentin zone was much wider; the apices were more open; calcification was arrested. The teeth of the rats killed at the end of 150 days of experimental feeding displayed lesions similar to those that we have reported in previous work (2). Changes observed in the pulps included engorgement, irregularity of the odontoblasts and degeneration, fibrosis and some calcification in the substance of the tissue. The predentin zone was greatly exaggerated and uneven. Strands of pulpal tissue could be found caught in the predentin and dentin. The dentin was poorly calcified, presenting alternate layers of light and dark staining tissue parallel to the dento-enamel junction. Necrotic pulpal tissue could be

found in the substance of the dentin. Apparently, the rachitis was so severe at the earlier part of the experiment that part of the pulp had become necrotic and was subsequently calcified.

Group II was on the same high calcium low phosphorus diet (A) as the preceding group but in addition received daily injections of parathormone. A study of the teeth of these animals revealed more advanced disturbances of the pulp and calcified tissues. The pulps were fibrotic and engorged, the odontoblasts were irregular and degenerated. The teeth and jaw bones were even less calcified than those of Group 1. The daily injection of small doses of parathormone into these animals on a high calcium diet seemed to accentuate the rachitogenic manifestations in the teeth and jaw bones, but did not fundamentally alter the character of the pathology.

Fig. 1. Rat fed Diet B (low calcium, high phosphorus) for only two weeks. Note caries of dentin—A, enamel lamella—B, and broad predentin layer—C. 16 mm. obj.; 4 × occular; orig. magnif. 150 ×.

Diet B was high in phosphorus, low in calcium and deficient in vitamin D was fed to Group III. All of the animals of this group displayed advanced dental disturbances. Even the teeth of the rat killed at the end of two weeks presented advanced pulpal pathology and incipient caries (*fig. 1*). The pulps were engorged and fibrosed. The teeth and jaw bones were much less calcified than those of the preceding groups. The 150 day animals in this group showed marked changes in the pulps. The odontoblasts were degenerated, the reticular cells assumed the characteristics of fibroblasts, the pulps were engorged; large calcified masses were found in the pulp, probably calcified necrotic tissue. The dentin and bone were very poorly calcified.

Group IV received the same daily dose of parathormone as Group II. These animals, however, were on diet B which was low in calcium and high in phosphorus. The animal sacrificed at the end of two weeks presented a histologic picture similar to its rachitic con-

Fig. 2. Molar of rat fed rachitogenic diet B (low calcium and high phosphorus) and receiving parathormone for only two weeks. Incipient caries of dentin—A, broad predentin zone—B, fibrotic pulp—C. 16 mm. obj.; 4 × occular; original magnif. 150 ×.

Fig. 3. Rat fed Diet B and receiving parathormone for 150 days. Note thinness of dentin—A, large predentin zone—B, and necrotic pulp—C. 16 mm. obj.; 4 × occular; original magnif. 150 ×.

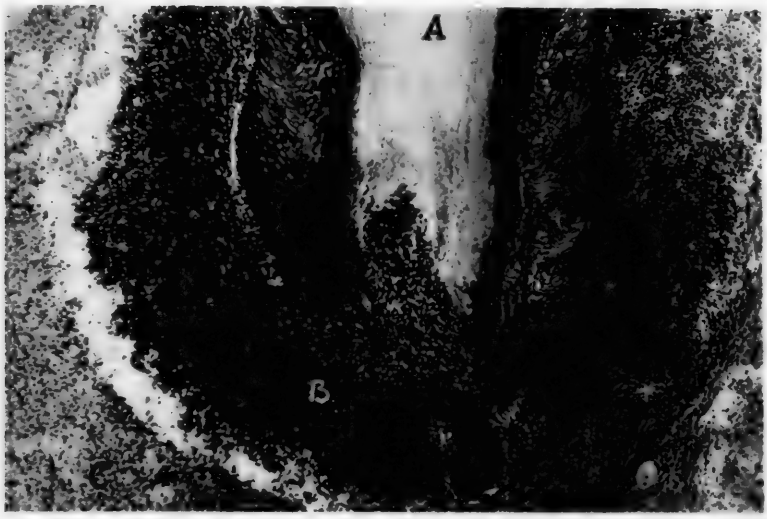

Fig. 4. Apex of molar of rat. See *fig. 3.* Necrotic pulp—A, periapical abscess—B, granulation tissue with dense leucocytic infiltration—C. 16 mm. obj. 4 × occular orig. magnif. 150 ×.

Fig. 5. Molar of rat fed Diet C (normal) for two weeks. No pathology can be seen. (Compare with *figs. 1, 2, 3*). Dentin—A, predentin zone—B, odontoblastic layer—C, pulp—D. 16 mm. obj.; 4 × occular; orig. magnif. 150 ×.

trol in Group III which received no parathormone (*fig 2*). The pulps were pathologic; the bones and teeth were poorly calcified; incipient caries was found. In the animals sacrificed at the end of 150 days, the pulps of the teeth were found to be appreciably more affected than those of the uninjected animals on the same diet. Inclusions of necrotic and subsequently calcified portions of pupal tissue were found in the substance of the dentin. The pulp chambers were comparatively larger in the teeth of the animals of Groups III and IV. The dentin was very thin in the majority of specimens in Group IV, the pulps of some teeth were so affected that dentin formation ceased in the early part of the experiment (*fig. 3*) and the apices remained wide open. Calcification of the jaw bones was also extremely poor. The degeneration of the pulp had gone so far in some

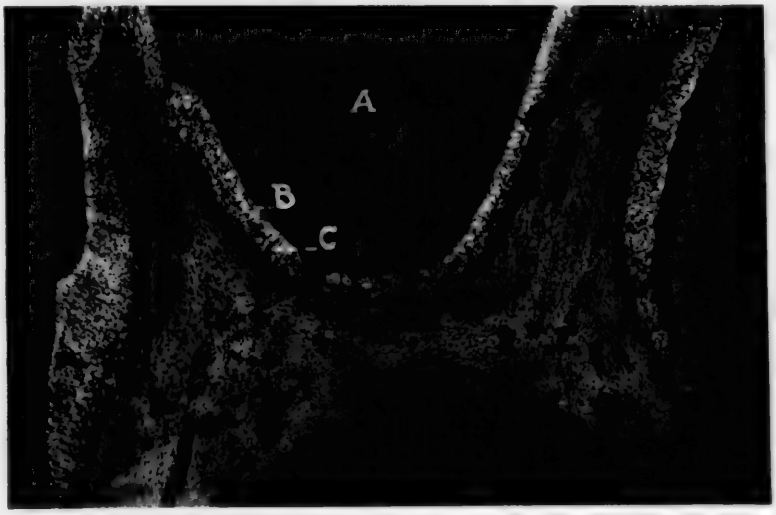

Fig. 6. Molar of rat fed Diet C (normal) and receiving parathormone injections for only two weeks. Dentin—A, predentin—B, odontoblastic degeneration—C, proliferation of peripheral connective tissue—D, diffuse fibrosis—E. 16 mm. obj.; 4 × occular; orig. magnif. 150 ×.

animals, that the pulps were completely necrotic and periapical abscesses developed (*fig. 4*). In the teeth whose pulps had become completely necrotic, no caries was observed.

Group V was the control group, receiving Diet C which is sufficient in mineral balance and vitamin content. The animals constituting this group although they were of the same age and litter-mates of animals in the other five groups, showed no demonstrable dental pathology (*fig. 5*). The pathology described in groups I, II, III, IV is in extreme contrast to the histologic appearance of the teeth and jaws of the normal control animals.

Group VI was also fed the normal diet, (Diet C), but in addition received daily injections of parathormone. At the end of two weeks of parathormone administration, very definite dental abnormalities were found. The odontoblasts were undergoing serous

degeneration presenting the characteristic "foam cell" appearance, (4) (*fig. 6*). The peripheral reticular cells adjacent to the layer of Weil had undergone fibrosis forming a distinct demarcation between the odontoblastic zone and the underlying tissue. The remaining pulpal tissue had also undergone a diffuse fibrotic change. In spite of this apparent degenerative process in the pulp, calcification of the dentin was well advanced. Only traces of predentin could be found. In the crowns and roots of the molars there was a suggestion of greater thickness of the dentin wall as compared to the control animal. In the incisor however, the predentin was unquestionably thinner and the dentin wall was thicker than that of the control animal. Rats seem to develop an immunity to repeated doses of parathormone.

The animals of the group sacrificed at 150 days did not show as marked a variation from the normal as was found at the end of only 14 days. The ability of the pulp to overcome degenerative processes occurring during earlier life is apparent. The contour lines in the dentin were exaggerated in comparison to the normal. Several alternate light and dark bands could be seen adjacent to the predentin. In many cases the amount of predentin was increased slightly. In these areas, several odontoblasts could be found imprisoned in the tissue. In two instances incipient caries was present in this group.

From the above study, it is apparent that although the rat is considered to be comparatively immune to parathormone, repeated injections of small doses do elicit a reaction in the dental tissues. The reaction varies with the amount of calcium and phosphorus in the diet. In our experimental work we found that any agents that affect the calcium-phosphorus metabolism will have a definite effect on the pulp and calcified structures of the tooth (1, 2, 3). Not only does the tooth proper become affected, but the periodontal tissues very frequently show marked abnormalities to be described in a future communication. Parathormone, as a potent factor in altering calcium metabolism in the animal body, does produce detectable changes in the dental tissues. In Groups I, II, III and IV, the rachitis has so affected the teeth of these animals that the effects of the parathormone were partially obscured. The pulps of the teeth of these four groups, killed at the end of two weeks, presented a similar histologic picture. In the animals sacrificed at 150 days an appreciable difference was found. In the teeth of the injected animals on high and low calcium diets, the calcification was more disturbed; reaction was greater than in the uninjected controls. The injection of parathormone seemed to accentuate the effects of the diets low in calcium and high in phosphorus, and high in calcium and low in phosphorus. The caries incidence in Group III was the highest; five of the six animals developed caries. The importance of calcium metabolism in the development of caries

in the rat is exhibited by the fact that both of the injected and non-injected animals of Group III and IV, killed at the end of only two weeks of feeding a diet low in calcium and high in phosphorus developed incipient caries (figs. 1 and 2). The effects of small doses of parathormone on the young rat fed a normal diet are much more apparent. At the end of the two week period a distinct and unquestionable pulpal pathology had developed. These lesions compare with those obtained after feeding a rachitogenic diet for 25 days (1).

Contrasting Groups I and III with Group V reveals the unquestionable reaction of the teeth of rats to calcium deficient, phosphorus deficient, and vitamin D deficient diets. Group V was fed a diet sufficient in calcium, phosphorus and vitamin D. Of the rachitic groups, the animals fed diet B were more affected. All of the rats in this group showed marked tooth and bone pathology; the pulps were markedly degenerated; much intra-pulpal calcification was evident; the predentin zone was very broad; the dentin was very thin and poorly calcified. Even the animal sacrificed at the end of two weeks showed well developed tooth pathology affecting the pulp, calcification of bone and tooth, and also presenting incipient caries affecting the enamel and dentin (*fig. 1*). Group I, fed a high calcium, low phosphorus diet presented marked changes in the pulp, dentin and bone. The pathologic findings were not as extensive as those in Group III. Group V was the control group. All of the bones and teeth of this group were normal (*fig. 5*).

Of the animals injected with parathormone, the two week Group VI specimen showed the most obvious changes. This does not indicate that the hormone does not affect or has less effect on the teeth of the animals in the other groups. The pulps of the animals receiving deficient diets are so greatly affected by the dietary alteration of calcium metabolism that the synergistic action of parathormone is partially obscured. The teeth and jaw bones of Group II were distinctly more affected than those in Group I which received no parathormone. Many of the pulps in the low calcium group had become necrotic (figs. 2 and 3) and periapical abscesses developed (*fig. 4*). It is probably this intense pulpal reaction that completely arrested calcification. At 150 days, the animals of Group VI did not show as marked pulpal disturbance as the two week specimens. Apparently

the animals had developed a resistance to the hormone. The injection of twice the usual dose (0.2 unit per 20 Gm. body weight) during the latter part of the experiment elicited very little, if any response.

Shibata (14), in 1929, produced lesions in rats teeth which were similar to human caries, by feeding diets which were rich in sugars, deficient in many respects and which contained large sized hard food particles. In spite of the multiple deficiencies, it is considered by some that the large particles were the causative factor. Since then the importance of particle-size in caries-producing diets has been receiving much attention. Hoppert, Weber and Canniff (15) reported that in the case of rats which received the commercially ground, fairly coarse corn meal, impactions of corn meal particles occurred usually in the lower molars. In these rats, they found, that "caries was initiated in about 8 weeks and at 6 months practically all the lower molars were involved in extensive decay." But when corn meal passing through a 60 mesh screen was substituted in place of the coarse particles of corn, there was no evidence of decay even at six months. These findings were confirmed by many other investigators. Although these later workers place special emphasis on particle size as the primary cause of dental caries, they do not entirely rule out the influence which is exerted by disturbed Ca-P metabolism. Thus Rosebury and Karshan (16) in discussing their findings with large particles state "On the adequate diets the incidence values (of dental caries) are in general lower; whereas on the deficient diets, the lesions were generally more advanced." This they interpret as being an effect of retardation rather than of prevention of dental caries. They state further "The reduced or delayed incidence of caries on adequate diets, as compared with deficient diets, on the other hand, suggests that dietary deficiency, although not a determining factor in etiology may increase the rate of the caries process perhaps by increasing the permeability of the defectively calcified teeth." In our experiments since the size of the particles was the same in all of the diets, the changes found in the different groups can not be ascribed to this constant factor. Although the ground corn used in preparing each of the diets contained some large particles, we found no caries in any of the rats which received the normal diets and whose calcium-phosphorus metabolism was not disturbed by the administration of parathyroid extract.

SUMMARY

Although the rat is comparatively resistant to parathormone, repeated small doses produced a distinct effect on the teeth and jaw bones. The dental pathology was similar in character to that found in animals whose calcium-phosphorus metabolism was disturbed by other means such as faulty diet, and calcium-phosphorus mobilization by excessive amounts of viosterol.

Diets, which altered the calcium-phosphorus metabolism, augmented the pathological manifestations produced by parathormone. In the animals receiving complete diets the pathology produced by parathormone was most obvious. In the dietary deficient groups, the dental abnormalities produced by the dietary calcium-phosphorus unbalance partially masked the similar changes produced by parathormone. In these groups, however, although the reactions were similar in character, the lesions were much more advanced.

The findings demonstrate the importance of the relationship of endocrine activity of the parathyroid glands to the dental structures, by influencing the inorganic salt metabolism.

We wish to express our gratitude to Dr. C. C. Lieb for his helpful guidance, to Dr. C. F. Bodecker for his valuable assistance in the interpretation of our finding, and to Dr. E. Applebaum for assistance in preparing the photomicrographs.

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CRANIAL STRUCTURAL VARIATIONS AND THEIR ASSOCIATION WITH DENTAL CONDITIONS

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Caries and pyorrhea: These are formidable diseases which constantly demand the attention of the dental profession in striving for their solution. Like any other organic diseases, the causes for either or both of these conditions are too obscure to allow any definite conclusions. Many factors are involved which may be regarded as direct or indirect causes for these diseases; these may be inherent from within the body or acquired from without due to extraneous influences. The fact that there is no dominant factor which may be held responsible for caries of the teeth or pyorrhea alveolaris makes it necessary to differentiate between contact decay or incomplete formation of the enamel of the tooth (such as deep fissures, etc.) and chemical decalcification of its entire structure. A differentiation must also be established between the recession of the alveolar border and suppurative degeneration of its investing tissues. It is important that further consideration be given the problem of how and when these conditions first occurred. Are they phenomena of recent or ancient times? If these degenerative changes in the masticatory apparatus of recent man are attributed to advanced civilization, what are the dominating factors accounting for some individuals of the present time going through life comparatively free from such disorders in spite of adverse influences. Conversely, what explanation have we for the diverse pathologic conditions in ancient man who was not subjected to the vicissitudes experienced by man of today. In this connection it is important that corollary evidence be introduced indicating the contrasting pathologic conditions and the incidence of dental disorders prevailing in ancient and modern man. The purpose here is to treat the far-reaching vital factors that have ultimately shaped the present human dental mechanism.

The clinical manifestations of dental diseases present many perplexing problems. Few of the manifestations generally associated with degenerative changes of the teeth or investing tissues can be attributed to a specific cause. Although it is well established that dental complications are of prehistoric origin it is difficult, if not impossible, to state how or when these disturbances first appeared in man. That the earliest human inhabitants were subject to dental diseases can be ascertained from the study of skeletal material of early prehistoric man. This study will also disclose that such manifestations were not confined to any particular group of people but were more or less common in one form or another among prehistoric people regardless of geographic distribution.

Before entering into the discussion of the subject, it may be well to examine more closely the entire subject of dental disorders. Such a study must include either a specific type of disorder, prevalent in all instances, or distinct morphologic differences. If such differences did occur, in what respect did they vary? It is also important to know what constituted dental diseases in man of the past, the relative incidence of such disorders, and the comparisons of such diseases with dental conditions in modern man.

Recent investigators of dental disorders in contemporary man have attributed the causes to local or nutritional influences. If such observations are confined to the complexities faced by modern man, the emphasis on the above influences as potential contributing factors to the diseases cannot be over-rated. However, this would be invalid in the consideration of dental disorders in prehistoric and ancient man because of the immense differences prevailing in the physical and environmental influences of prehistoric and modern times. Whereas contemporary man is subjected to diverse and uncontrollable living conditions, life presented to his ancestor little variation, especially in his food habits or nutrition. If, on the other hand, like circumstances produce like results and if the physical and environmental conditions of prehistoric man were essentially alike, what was the cause for differences in the susceptibility to dental diseases at the very early periods of the human race, beginning approximately with the Neanderthal period. Since there is available skeletal evidence to indicate that our earliest predecessors were not totally free from dental disease,

it is imperative that we ascertain the differences as they occurred at the earliest stages of human history. From examination of skeletal remains of very early man it is evident that a prevailing variation, either in skeletal or dental structure, assumed definite proportions either in structure, shape, size, configuration or other distinct contrasting characteristics. It is necessary, therefore, to determine not only how or when dental disorders first appeared in man but whether structural variations could be associated with either normal or adverse dental conditions in the individual. Upon a reasonable assumption we may arbitrarily attempt to classify Homo Sapiens as belonging to three periods of human history (*fig. 1*). This classification may include the earliest known representatives who may be associated approximately with each given period, with varying pathologic manifestations of each assumed period. These peoples may be considered as belonging to archaic or prehistoric man, ancient and primitive man, and recent or contemporary man.

The earliest Neanderthal and Neanderthaloids offer a scant amount of skeletal material. Sufficient data can, however, be collected to ascertain the presence of degenerative changes affecting the alveolar process or the earliest manifestations of dental abnormalities in man of archaic time. From the evidence now available one may conclude that the breakdown of the investing tissue in Neanderthal man was mainly of the atrophic type with some suggestion of a suppurative condition manifested in some instances.[1] Visible signs of alveolar abscesses originating in the pulp of the tooth, unless due to trauma, doubtless resulted from extensive attrition caused by grit in the food. Except for the phenomenal Rhodesian man, who was the victim of every type of dental disease common today, caries among very early man perhaps was either rare or did not exist.

With the appearance of the Cro-magnon of southwestern Europe and of other prehistoric beings of corresponding periods, whether of African or Asiatic origin, the occurrences of alveoclasia became more apparent and were often accompanied by premature loss of teeth.

[1] References to Neanderthal skeletal material include only those known specimens which furnish either all or most of the skull, or complete maxillae. The other specimens, recently discovered, representing Homo Sapiens in earlier stages, consist chiefly of fragments or incomplete parts of the skull and are therefore of little clinical value.

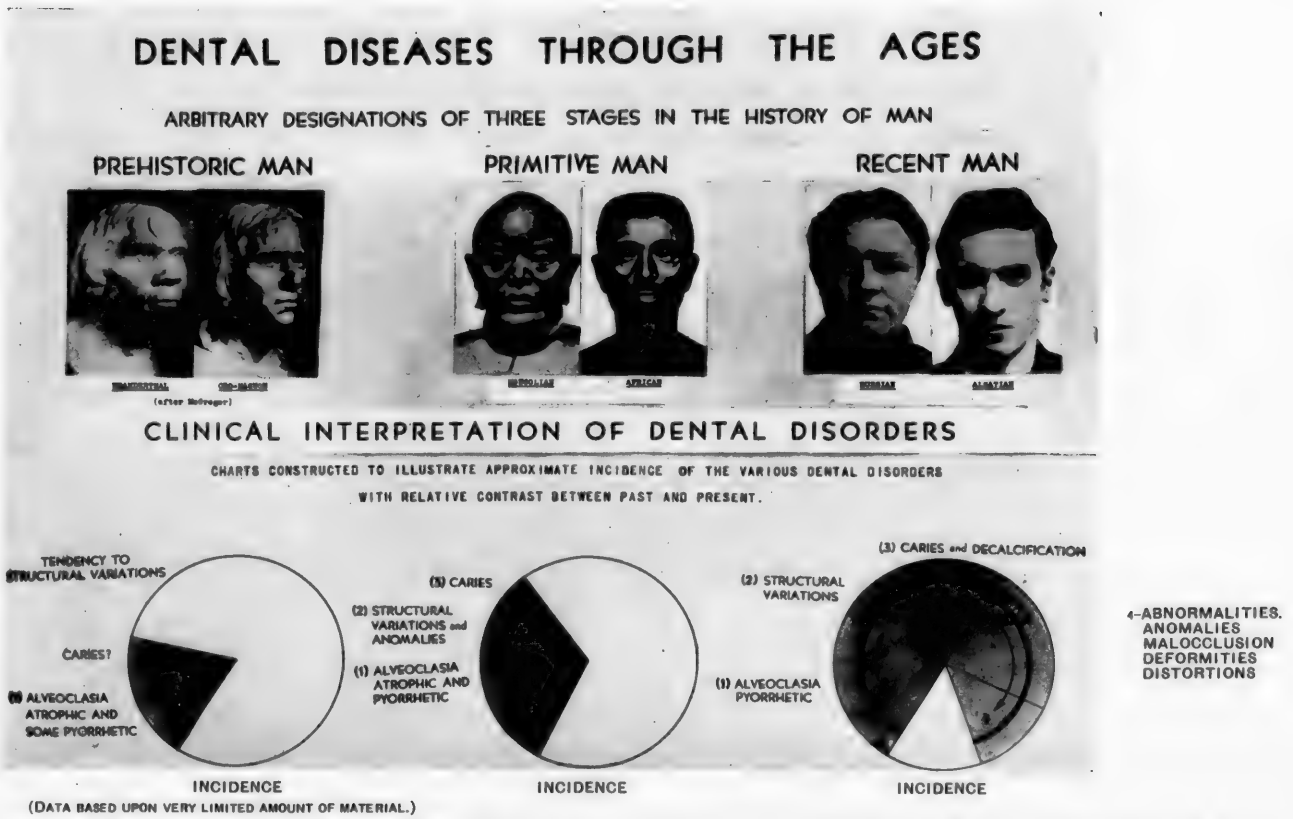


Fig. 1. This is a composite concept dividing Homo Sapiens into three periods each represented by two distinct contrasting types: 1. Reconstructed representatives (by McGregor) of prehistoric man. 2. Primitive (composite illustration). 3. Recent man. A schematic attempt is presented on a purely theoretical basis to illustrate the approximate state of dental conditions in each given period. The skeletal material of man of the early stone age is too limited to warrant any definite conclusions. The illustrations represent clinical interpretation of incidence to dental disorders from earliest known to recent man as revealed through skeletal and clinical investigations.

Among more recent man of ancient time, such as Egyptians and contemporary peoples, there are to be found varied types of dental disorders closely resembling those common in man of today. Dental aberrations and anomalies and variations in the shape of the maxillary arches and patterns of teeth commonly occurred among the ancients of African and Asiatic origin. These phenomena, moreover, assumed further alteration and the incidence of caries became more apparent. In the course of time there appeared a greater degree of degeneration of the teeth and investing tissues and a pronounced departure from the original structural configuration of the dental apparatus became more evident.

Comparison of dental disorders from the earliest known specimens to modern man reveals a wide range of differences in the incidence, degree, and morphology of the disorders. It is to be observed that, even among earliest man, in the instance of alveolar involvements there existed a distinct morphologic difference in the type of alveoclasia. From earliest time, and throughout succeeding ages, two general pathologic trends in alveoclasia are perceptible: one pyorrhetic and the other atrophic. It would appear that some individuals enjoyed, in early life, a relative immunity to alveolar diseases as indicated by the fact that a gradual atrophy of the alveolar border is evident, which no doubt appears only in later stages of life and may be attributed to the normal course of advancing age.[2] In the pyorrhetic type the extent of the pathologic process showed considerable variation; in some instances it was confined to a few teeth only while in others the entire alveolar process of both jaws was involved. It is also significant that, unlike atrophy of the bone which usually occurs with the advance of age, the suppurative type may in early middle age develop complications that frequently result in premature loss of the teeth. It is in the suppurative type that the tuberosities and ridges of the alveolar process become totally absorbed following the

[2] Even these natural phenomena of atrophy of the bone and loosening of the teeth, which accompany advancing age, display divergent tendencies in individuals at varied stages of life. It is obvious that all individuals do not follow the same course of physical and dental vigor through life, inasmuch as chronological age does not necessarily indicate a period of approaching senility or dental decline. Similarly, this dental decline in contemporary man very often precedes every other evidence of impending senility and may even occur during the period that we consider prior to middle age.

loss of the teeth. What may be regarded as most significant is the curious phenomenon whereby the investing tissues suffer from cellular insufficiency or hypocalcemia of the bony matrix of the alveolar process; yet the teeth in these instances are usually free from degenerative changes such as caries or decalcification.

There is an exceptional advantage in studying structural variations in the bone formation, as well as the gross morbid anatomy, and their associations with dental disorders which are more perceptible in anatomic dry specimens than in clinical observations. This method of study discloses the variability in the thickness of the external alveolar plate covering the roots, not always obvious clinically. Further, it will reveal that in proportion to the size, shape, thickness and compactness of the walls of the skull there is invariably a proportionate increase or decrease in the thickness and density of the bony matrix of the alveolar process. The degree of variation ranges correspondingly from a uniformly thick reinforcement, involving the entire border of the alveolar process, to an uneven and thin distribution of the protective layer of bone barely covering the roots and very often partially exposing them. (*The latter phase should be of interest to the specialist treating alveolar diseases, since the resorption of bone is not self-limiting in the pyorrhetic type and will continue to break down in spite of treatment*) (*figs. 2* and *2a*). While the function of bone growth is cellular proliferation, which is influenced by internal and extraneous forces, the usual tendencies are for the structural formation as well as the degenerative changes to follow a congenital pattern throughout the life of the individual. The above phenomena may be observed in the skeletal remains of ancient man but are much more prevalent in recent man and the lower animals, particularly those of the primate family in which the degree of dental pathology ranks next to the human. In this connection one should note the craniologic diversity in the structure of the heads as well as other variations which also prevail in the respective groups of primates.

Based upon the above observations related to the prevailing variability in the bony structure of the alveolar process in different individuals an approach can be made to consideration of the various phases associated with the whole dental problem. Clinical experience, as well as study of skeletal material, reasonably establishes the associa-

tion of predisposition or immunity to dental diseases with pronounced physical and structural characteristics of the individual. When this phenomenon is referred to recent man, certain indisputable criteria must be recognized as bearing upon the causes for the marked decline in present human dentition. The dental condition of contemporary man is too obvious. He not only has fallen heir to the dental disharmonies of his predecessors but he has as well developed a dentition including a wide range of structural variations and a predisposition to dental disease unparalleled in human history. In a general sense the manifestations of dental disorders are precisely attributed to many

Fig. 2. Anatomic laboratory specimens. Sections of mandible indicating variability in the bone structure which may point to the innate predisposition or relative immunity to alveolar disturbances. (Courtesy Medical College, University of Cincinnati.)

outstanding causes, e.g. heredity, adaptive food habits, poor nutrition, the influence of the secretory glands, environment, and social and economic factors. Analyzing further some of these factors at fault we consider the physical and local correlation of organisms, the Lamarckian interpretations of the use and disuse of organs, and the laws of Darwin relative to the adaptation of specialized organs in the course of natural selection. Acquired characteristics, mutation, and mixed matings obviously account for further radical body alterations and physical transformations. The foregoing factors, therefore, constitute a logical basis for the study of dental pathology (*fig. 3*).

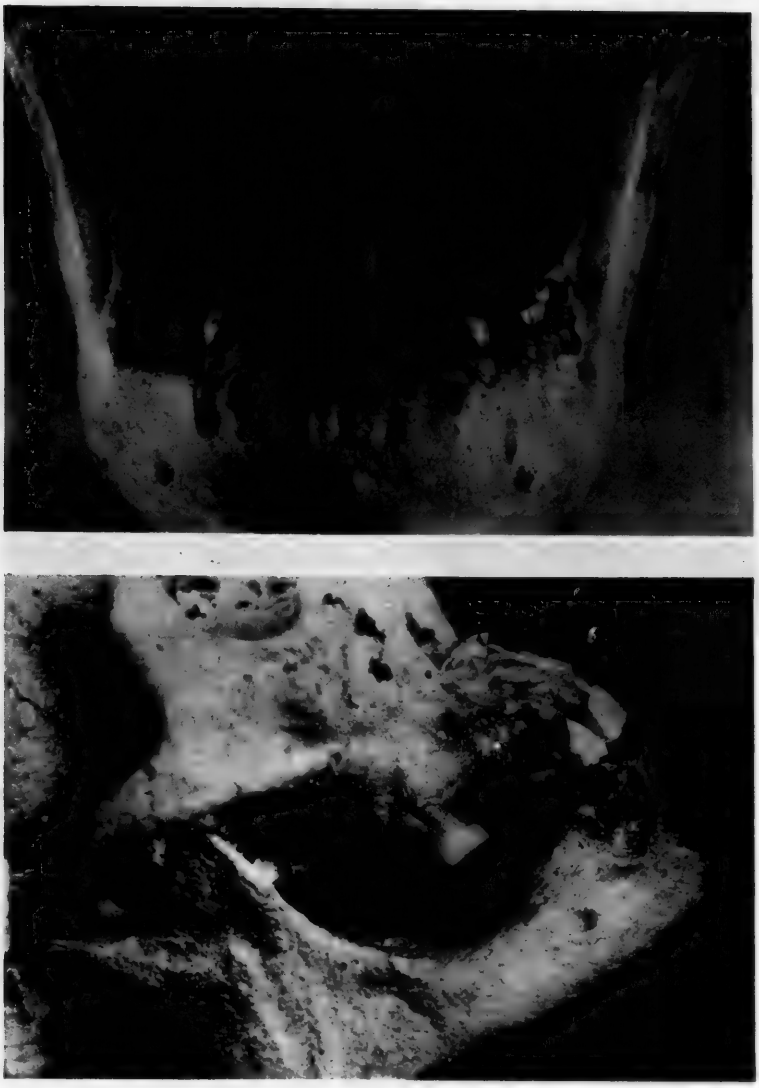


Fig. 2A. Anatomic Specimens. Cellular degeneration of the alveolar processes, both of which illustrate bone disintegration usually associated with pyorrhea. (Courtesy Medical College, University of Cincinnati.)

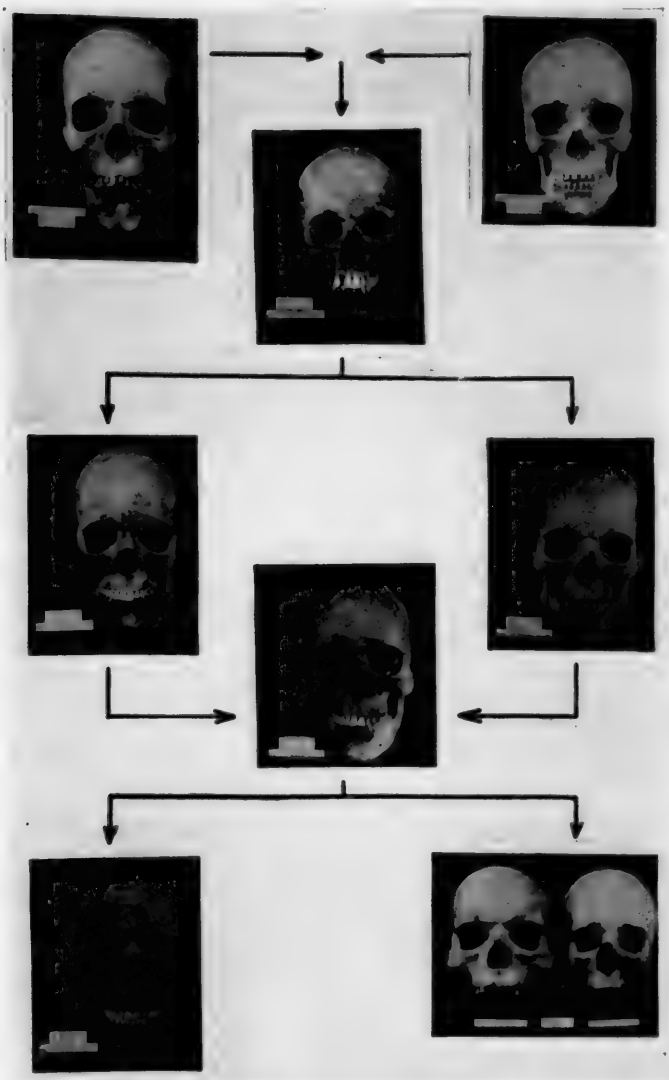
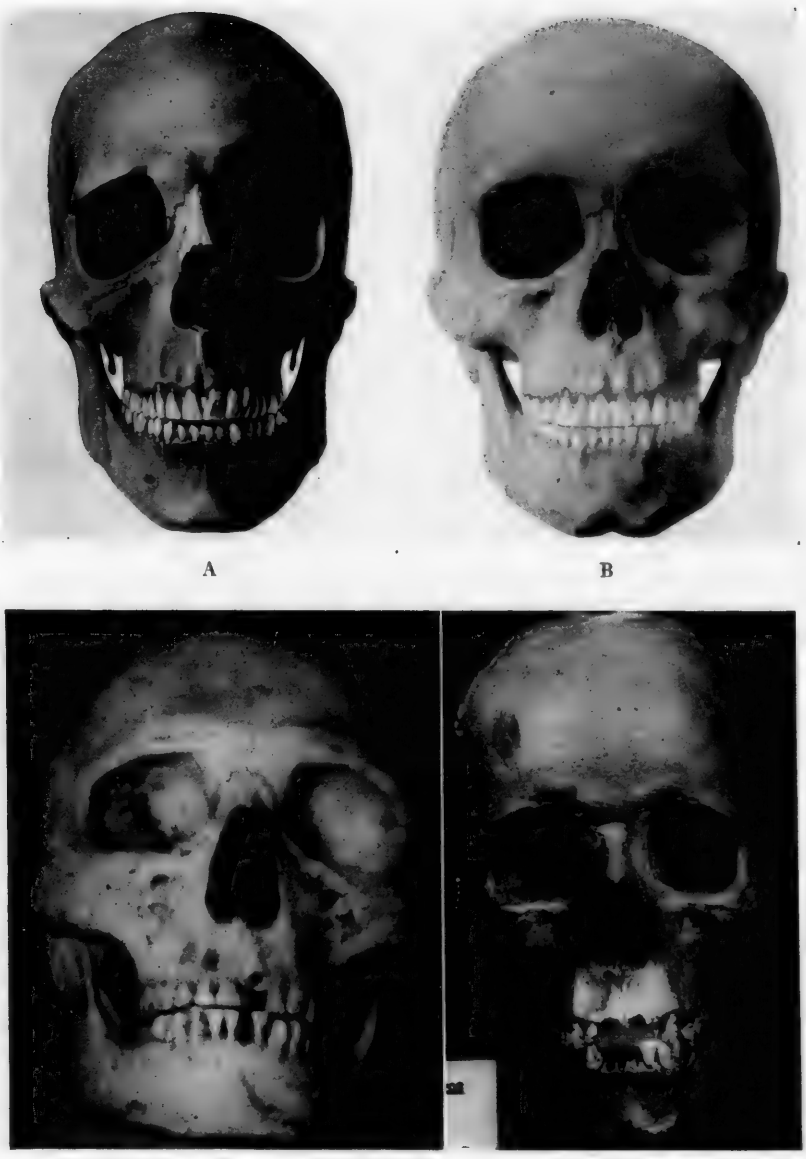


Fig. 3. Blending of types results in departure from original characteristics and constancy, and in formation of a new type different in form and cranial and dental structure. These skulls are those of an ancient people (Persians) belonging to the same region, dating 3500–1500 B.C. (Courtesy—University of Pennsylvania and Dr. Wilton Marion Krogman.)



A B C D

Fig. 4. The (so-called) long and narrow-headed (A), short and round-headed (B) original types, both of which normally retain an ideal dental condition. Typical Mongolian and African skulls; recent Mongolian Urga region (C) (Smithsonian Museum); Egyptian (D), B.C. 2500 (Hammann Museum, Western Reserve).

Whether the early human prototype hailed from a common ancestor and has spread over all parts of the globe or has evolved from different branches of the humanoid tree, he has, nevertheless, introduced distinct examples of physical differentiation. According to recent accounts, East African prehistoric man (described by Leaky and associates [15])[3] characteristically follows the structural variability prevailing in the prototype of the Cro-Magnon of southwestern Europe. Some specimens among the East Africans, but of a later period, also include an inhabitant who has diverged from his earlier ancestors and who more closely resembles recent man. Whatever the circumstances have been, possibly the resulting adaptive accommodations to special food habits or other factors influencing structural changes, the newcomer was obviously representative of a changing strain of cranial modifications. Perhaps for the same reason of racial diffusion, environmental adaptations, or the origin of separate genera similar characteristics are observable among the primitive and indigenous peoples of the most ancient times. Corresponding closely to the respective physical structures of the body, the skulls were patterned according to distinct shapes; long-headed, short and round-headed, coffin shaped from top down, or conversely tapering from the widest portions of the angles of the mandible to the top of the cranium. Further amalgamation resulted in constant structural modification which doubtless followed with the multiplication of the human race. Invariably, however, references are made to the short-headed Mongolian or to the long-headed African as the representatives of the two distinct ancient types (*fig. 4*).

Though nothing specific can be pointed to as having given rise to the phenomenal physical divergence, some definite variations developed in the shape and structure of the dental arches, palates, and mandibles. Palates and dental arches were sometimes wide or narrow, high or low; while mandibles varied in depth and thickness. The size, shape, and pattern of the teeth corresponded proportionally either to the original type or conformed in their aberrations to the acquired amalgamations. The common occurrence of such a wide range of variation prevailing in ancient and primitive man is revealed in the skeletal remains of the East African (15), early Egyptians,

[3] Specimens at Royal College, London.

Persians[4] (Tepe Hissar—3500 to 1500 B.C.), American Indians, South Americans, Pacific Islanders, and many other prehistoric peoples. In more recent primitive man, the Eskimo is an exception, an excessive use of the jaws being evident. In many instances, among the Eskimos, a disproportional compensating hypertrophy is especially notice-

A B

C

Fig. 5. Contrasting structural and dental conditions as revealed in these specimens, representing distinct types of health, vigor and debility. Especially skull C in which extensive atrophy and dental disorders resulted in complete resorption of the tuberosities and perforation of the floor of the right antrum. All these specimens are from the same region, Egypt—period—12th Dynasty. (Courtesy—Smithsonian Institute.)

able in the thickness of the mandible. In all other cases, alterations, when they occurred, often led to structural decline. Reasonable consideration should be given to such aberrations when they are accom-

[4] From the Museum of the University of Pennsylvania. A complete and detailed account of general and dental findings will appear from other sources.

panied by hypocalcification of the bony structures which may have been influenced by lack of function, faulty metabolism, or environmental adaptation. Nevertheless, a primordial tendency has been perpetuated in some cases in recent man. This type apparently has not been affected by the same environment or adaptive accommodations which gave rise to the prevalent degenerative occurrences. In either instance this unique phenomenon persisted regardless of time and geographic distribution as evidence reveals similar diversified characteristics prevailing universally notwithstanding a similarity of adaptive accommodations and of food habits. Contrasting features, when they occurred, either in the preserved type or in the modified form, were followed by a corresponding conformity either to the original type or to sequent alterations in the masticatory mechanism. Perhaps these two factors may account for the selective immunity or the predisposition to dental disorders as they are usually associated with the specific structures of the individual and the corresponding dental conditions. From the accumulated evidence *it is invariably possible to correlate sturdy jaws and relatively normal teeth with the sturdy individual in contrast to the decrepit dental condition of his fellow men* (*fig. 5*). In such isolated instances where tendency toward perpetuation of original physical ruggedness or stalwartness is still in evidence, clinical as well as skeletal examinations reveal correspondingly favorable dental conditions. It is to be observed further that where the outstanding features closely border on the original homogeneity, this type, whether found in primitive man or recent man, is invariably endowed with sturdy jaws, well-formed and rounded U-shaped arches, short but well-developed teeth, and reciprocative reinforcement of the alveolar process. The contrasting type shows the counterpart of the conditions just described and may be said to represent a combination of altered modifications. Of these, in extreme instances, the dentition in modern man gives the appearance of rudimentary functionless organs. Instead of the thick, dense and compact bone of the walls filled with dense cancelous tissue as in the former type there is an assortment of thin, light-weight bone, sparsely filled cancelous structure and great variety in the pattern and size of the teeth and shape of the jaws. It may be stated conclusively that it is

the latter type which readily yields to degenerative changes of the dental organism both in tooth structure as well as alveolar support.

The question of why certain types show cumulative or sudden variations and others, regardless of adverse influences, retain for long periods their ontogenetic characteristics remains an open one. It is to be suspected that such variabilities may be a phylogenetic expression of the continuous changes in the teeth and jaws from the earliest human ancestry and that perhaps the dentition of man in the future will be different from that of man of today. This inference may be supported by observation of the pronounced tendency in the dentition of modern man toward a gradual reduction of the size of the teeth and jaws, the elimination of the third molars which are inclined to become vestigial organs), peg-shaped lateral incisors, congenitally missing and unerupted teeth, and the profound deterioration of the investing structures. These changes are not spontaneous occurrences. The significance of these phenomena lies in the fact that a pronounced series of transformations has, to a large extent, been in progress since Neolithic times. Nature herself lays no restriction on time; *unless man may henceforth be instructed in the acceleration of a more rapid radical transformation toward an ideal physical and dental homogenesis.*

For the present it is important to observe the validity of the laws of heredity and the impressiveness of mutation—provided a breach in the continuity of successive generations from the original stock is not interrupted by too wide a gap. The same principle applies to acquired characteristics where deviations and variations as well as susceptibility, at least to dental disorders, are transmissable. Both original and acquired characteristics are congenital in their origin and will continue their specific individual tendencies unless arrested by extraneous influences after birth.[5] The last phase to consider here is the clinical significance in which the structural characteristics influence correlation of organisms and where modification of one part involves modification of another. It is obvious that morbid anatomic alterations are not compatible with normal physiology. Irrespective of the cephalic index, whether in the long-headed or short-headed

[5] That physical characteristics are transmitted from parent to progeny is too obvious, and that distinguishing dental features often continue a hereditary tendency is borne out clinically.

person, the presence of radical deviations from normal anatomic arrangements will correspondingly influence physiologic functions.[6]

In the early days of human dominance, regardless of geographic localities, when craniologic division was based on either long-headedness or short-headedness, in most instances uniform sturdiness of the dental make-up existed. With the advance of civilization, however, the brachycephalic more frequently exhibits dominant structural characteristics while striking deviations become pronounced in the dolichocephalic type. Sturdy jaws with ideally rounded arches and well-developed teeth furnish the most obvious example of the more uniform dental complement frequently associated with the brachycephalic in modern man. The brachycephalic, which by some observers is considered an entity distinctly associated with the original characteristics of the genus Homo, is supposedly endowed with an ideal dentition reputedly free from dental disharmonies; a condition not prevalent in the dolichocephalic. This conception embraces many converging lines of evidence pointing to such traits as the origin in the brachycephalic of form which may appear more or less constant in structure. Clinical evidence sometimes compels the acceptance of a deduced association of short-headedness with immunity to dental disorders. While such interpretations are not to be given too prominent a significance they, nevertheless, suggest relative potentialities for immunity. But the lesser frequency of dental disease in this type is rather due to the uniformity in the anatomic structure of the masticatory mechanism.

The belief that the long-headed type is potentially predisposed to dental pathology is valid only when the otherwise normal individual exhibits complex distortion or deviations from natural coördinated development. One explanation may be the fact that modification of unfavorable conditions are more conspicuous in the long-headed type because of the crowding of the teeth in undersized jaws. This condition assumes disproportional prominence because of the underdeveloped jaws which either force the anterior teeth into protrusion or accentuate prognathism of either jaw. Wherever normal con-

[6] The term normal as applied here refers to the physical and dental constancy when predominating in ancient man from either African or Asiatic source or where alterations have not made spontaneous inroads.

ditions or original characteristics prevailed in the long-headed types, they, regardless of race, period or geographic distribution, were not affected by extreme alterations or dental abnormalities. Such uniformity is manifest in the normal shape and size of the dental arches and teeth as well as the depth of the jaws and the density of the alveolar bone supporting the teeth. The tendency to caries in this type is not as common as it is in those with constricted palates, deformed jaws, and crowded and malformed teeth. Decalcification of tooth structure as it occurs in modern man spares neither type, whether short of long-headed. This may be attributed to extraneous influences, irregularities in function of the internal secretory glands, undernourishment, and malnourishment.

Alveoclasia or recession of the alveolar border in the structurally normal type seldom occurs before middle age. It is usually of the atrophic variety and the degree of bone destruction is, to a large extent, restricted due to the thick layers of compact bone of the alveolar walls supported by dense cancelous tissue and nourished by a well developed Haversian system (*fig. 6 & 6a*). Moreover, shrinkage of the jaws in the atrophic type is usually self-limited, following the loss of the teeth, and seldom results in the distortion of the facial contour. In striking contrast, in the other type which from an early period experienced continuous alterations resulting in cumulative dental diseases there are found the effects of morbid anatomy and depressed physiology leading to irreparable local and perhaps constitutional damage. It is this type which is more susceptible to the ravages of caries and decalcification of the teeth. Likewise, the ill-deposited bony matrix of the investing structure yields to degeneration and is usually accompanied by suppuration or pyorrhea. Even after the teeth are lost cellular disintegration continues and often culminates in the complete absorption of the alveolar ridges. The early development of a phenomenal thinness in the palate bone and of the accessory sinuses and the accompanying failure to benefit from cellular proliferation during body growth make for ready predispositions to decline and injury (*fig. 7*).

From the foregoing observations, and from others not included here but available for study, the evidence is conclusive that structural variations or departures from normal types are overabundant. Clini-

Fig. 6. Anatomic dry specimens depicting variations in shape, size, thickness, and uniformity of bone, and respective configuration of skulls. These distinctions are also evident in the dental make-up, corresponding respectively to normal or abnormal conditions. (Courtesy Medical College, University of Cincinnati.)

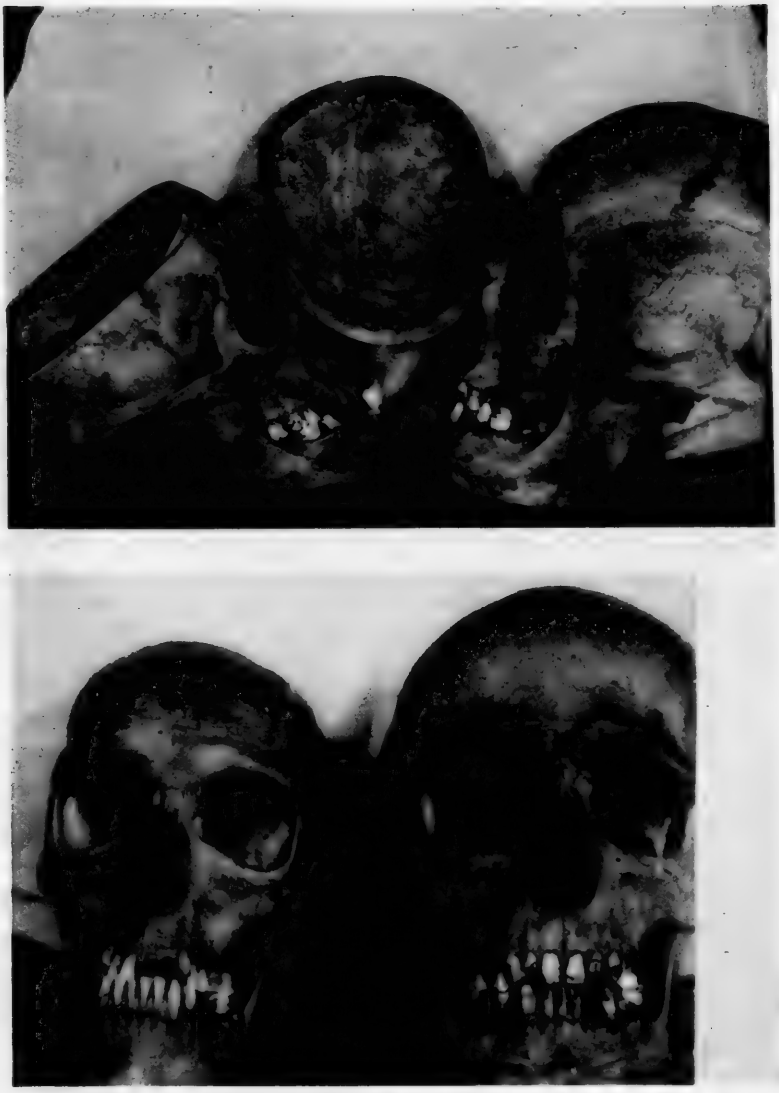


Fig. 6A. Structural variations in these anatomic dry specimens reveal not only existing dissimilarities in size and formation of bone, but relative association of dental conditions with each type, or inherent physical features. (Courtesy Medical College, University of Cincinnati.)

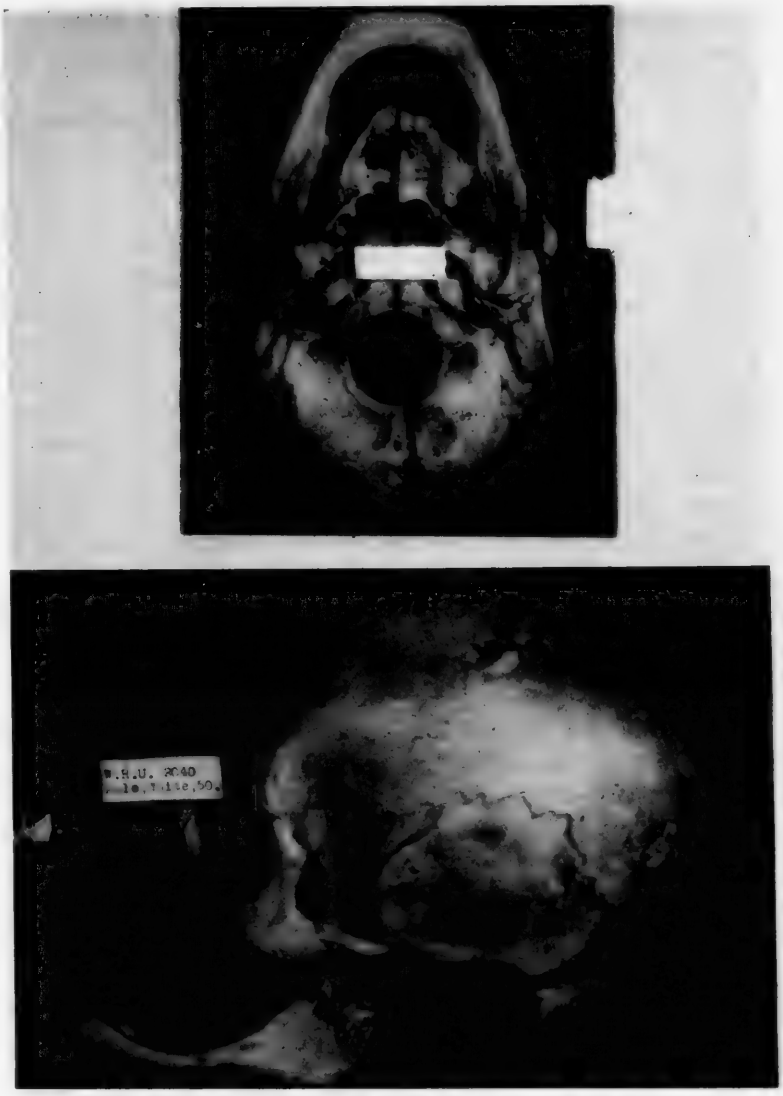


Fig. 7. Male, white age 50. Final stage in retrogression of human masticatory mechanism. Premature loss of teeth and alveolar process corresponds to structural conditions of the skull. (Courtesy Hammann Museum. Western Reserve, Cleveland, Ohio.)

cally it is also obvious that the altered type displays a greater tendency toward irregularities or abnormalities. These physical factors when further complicated by extraneous influences and especially by incompatibilities to normal living conditions would seem to indicate the constancy and tenure of the human masticatory mechanism to be in a highly precarious state. Physical changes and variations in the structure of the skull have occurred from the earliest known origin of man to recent time. These changes have a marked influence on the dental organism, which exhibits diverse tendencies associated with predisposition or immunity to dental disorders (*fig. 8*). The causes may be the result of natural disharmonies or acquired characteristics, environmental adaptations, heredity, mutations and transmutations, disfunction of the ductless glands, faulty food habits, nutritional influences, imprudent mating of the physically unfit, and generally a lack of the essentials for normal life.

The study of skeletal material reveals profound variability and structural alterations in prehistoric and primitive man and clinical observations give credence to the same state in present day man. The distinctly contrasting types, approximating either normal or altered, are of congenital nature unless the body development is influenced by some radical changes in early life. Genetically, the teeth and jaws retain the physical and anatomic properties specific to the individual and departure from physical constancy and physiologic requirements usually leads to alterations of the dental apparatus which is subject to degenerative changes. The basic features retaining specific identity of the original type of early man are the large skull, broad and fully rounded dental arches, deep and heavy mandibles, thick and compact bone, and sturdy masticatory apparatus with a lesser tendency to dental diseases. The light-weight thin bone, irregular shaped jaws, distorted arches and teeth are the common features of the contrasting type which readily yields to dental complications.

As for the future status of dental conditions in man, it is, for the present, hazardous to predict. Much depends upon the physical well-being of the human race and environmental accommodations necessary to normal life and health. In this connection it may be suggested that man of today can help himself by devising means for reestablishing a more normal physical and dental homogenesis through

conservation of human energies and reassertion of his appreciation of the meaning of life and health. Further, what may appear as a logical deduction is that, unless deviating modifications revert to some of the original characteristics through the possible law of mutation, a protracted departure from the original stock will never regain its lost structural characteristics.

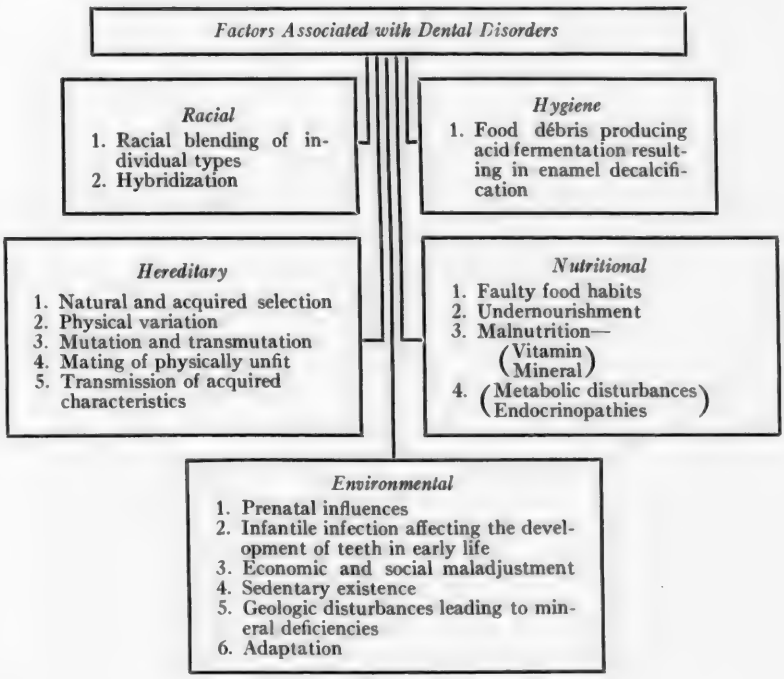


Fig. 8

The author wishes to express his appreciation to the Hamann Museum, Cleveland, T. Wingate Todd, Director; American Museum of Natural History, New York City; Smithsonian Institute, Washington, Ales Hrdlicka, Director; College of Medicine, University of Cincinnati; University of Pennsylvania, Philadelphia (Dr. W. M. Krogman), Royal College, London, Dr. Leaky's Collection; and Donald Dickson, Lewiston, Ill., and many others for the opportunity of examining their collections of skulls.

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ABNORMALITIES ENCOUNTERED IN DISSECTION OF THE HEAD AND NECK OF AN ANENCEPHALIC MONSTER

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Figs. 1 and *2* indicate the anthropoid appearance of the face due to exophthalmus, the absence of forehead and long upper lip. At the right corner of the mouth is a villus (1), (not seen in the photograph; see *fig. 3, c*). The neck is obliterated, the chin and chest lying in the same plane (*fig. 2*). The head is sharply thrown back and from the dorsal aspect the bifurcation of the spinal cord is seen to expose the meninges as well as spinal nerves, some of which present ganglia. The dorsal view of the head presented a bluish fibrous tissue flap, transparent when reflected, which showed the calvarium to be absent, and exposed the superior body surface of the *os sphenoidale*. Hypophysis and internal carotid vessels were identified. In the neighboring tissue were the trunks of the *nervus trigeminus*, not identified until traced by dissection, as the Gasserian ganglion was absent and the petrous portion of the temporal bone ill-defined. The presence of pituitary gland is contrary to the findings of Browne (2), who states "that the characteristic physical signs of anencephaly apart from the absence of the brain and cranial vault, are connected with the absence of the pituitary gland."

The absence of a brain classified the monster as anencephalic, the bifurcation of the vertebral column with its meningeal protrusion identified it as a spina bifida, and the opening existing in the cephalic portion of the vertebral column determined the spina bifida as superior.

History. Mother, white, married, age—51 years, weight—140 lbs, height—5 feet, 4 inches; primipara. Reason for delivery, term; state of general health, good; conceived at onset of menopause; this onset was not marked by any constitutional symptoms. Edentulous. Attended prenatal clinic. Date of last menstrual epoch, intermittent; stated menstrual age, not reliable.

Anencephalic monster + spina bifica (superior). Condition of embryo, II (Streeter's Grade I, II, III). Sex, female, weight—813 grams.

The following report is based on dissection of the right side, as mutila-

Fig. 1

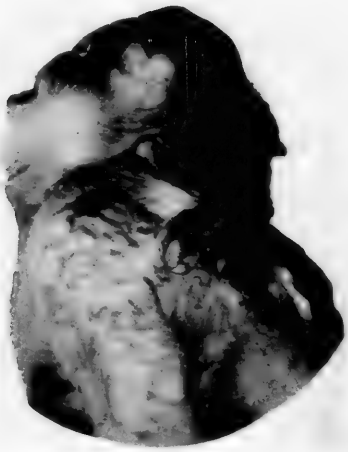

Fig. 2

Fig. 3

Fig. 4

Fig. 3. A—facial muscle group, B—platysma colli, C—villus.

Fig. 4. D—constructing fibers of capsule passing around parotid gland, E—branches of facial nerve, F—parotid gland.

tion of the left mandible was necessary for purpose of making histologic slides.

Outline of incision. 1. A median incision was made from supracranial crest to a point opposite the level of the right axilla. 2. From supracranial

crest an incision was made in a coronal direction to the pinna, continuing anterior to the latter to a point just below the lobule, then laterally to the

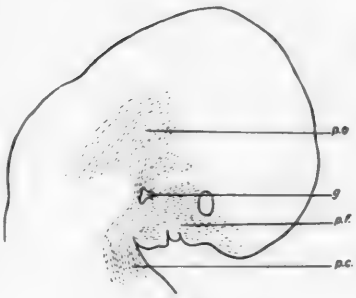

Fig. 5

Fig. 6

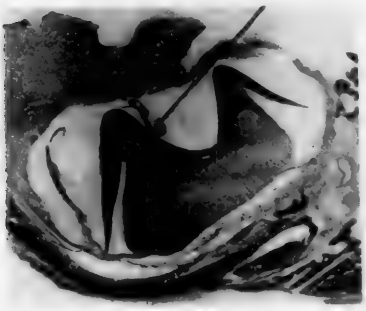

Fig. 7

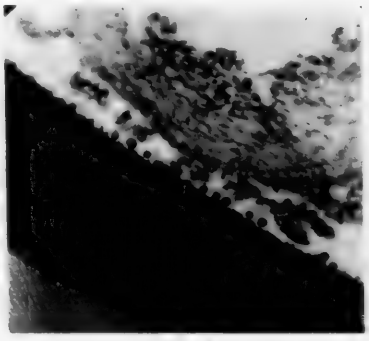

Fig. 8

Fig. 5. Superficial layer of platysma musculature of embryo. *p.o.*—platysma occipitale, *g*—oral fossa, *p.f.*—platysma faciei, *p.c.*—platysma colli. (From Keibel and Mall, Human Embryology, Vol. I, p. 513.)

Fig. 6. G—temporal muscle, H—parotid duct, I—masseter muscle.

Fig. 7. Section through left second molar. Arrow indicates area in *fig. 8*.

Fig. 8. Higher magnification of area in *fig. 7* showing calcospherites.

area of the acromium process of the clavicle, thence downward to the anterior border of the axilla (*fig. 3*). The resultant flap of skin was reflected downward.

Facial muscle group. The facial muscles, quadratus labii superioris, zygomaticus major, and caninus, were undifferentiated, consisting of a muscle band attached to the bony area lying between the root of the nose and the eye, extending laterally and downward, thus occupying a large portion of the face (*fig. 3, a*). This muscle tissue, like many of the muscles encountered, had a pitted appearance probably due in great measure to shrinkage as the result of fixation. Ramifying in this muscle band were branches of the n. facialis (*fig. 4, e*), one of which could be traced medially to its termination at what appeared to be the infra-orbital foramen, so that it corresponds to the infra-orbital branch of the seventh cranial nerve.

Platysma. Laterally this facial muscle group was attached to a band of fibers which extended from a pre-auricular position and curved downward under the chin to meet its fellow of the opposite side (*fig. 3, b*). The lower fibers of this muscle became confluent with those of the pectoralis major. It seems likely that this circular muscle band indicates a retention of the embryonic arrangement of the platysma muscle. A possible explanation is offered in the development of this musculature (3). "The entire facial musculature arises from the closely packed mesenchyme which fills the second branchial arch. . . . The main stem of the n. facialis passes into the arch" (9 mm stage) and ends in the premuscle tissue which is to form the entire musculature supplied by this nerve.

"This premuscle mass increases in size and in an embryo 13.7 mm. in length has begun to spread out not only ventralwards but also dorsalwards and aborally. *It also extends towards the shoulder* to form the platysma. . . . As the muscle rudiment spreads out, the n. facialis becomes divided into several branches which follow the wanderings of the muscle tissue. . . . In a 15.5 mm. embryo the platysma rudiment has extended orally over the hyoid arch, and caudally to the region of the sternum and shoulder girdle. It has pushed medially and begins to unite with its fellow of the opposite side [*fig. 5*]. The spreading of the muscle headwards takes place along two paths, separated by the anlage of the outer ear. . . . The facial portion of the platysma splits at the upper part of the neck into two layers, a superficial, . . . and a deep . . . one. Futamura designates the superficial layer as the platysma faciei and the deep layer as the sphincter colli. . . ."

Further differentiation of these muscle layers takes place, but enough has been quoted to warrant the assumption that this circular muscle group represents an embryonic arrangement of platysma fibers.

Reflection of the facial muscle band exposed all that was found of the parotid gland, resting on the masseter muscle. The gland resembled a

hemisphere with its convexity turned outward. It had a constricting fibrous attachment which arose from the side and root of the nose and passed circumferentially about the gland (*fig. 4, f, d*).

Removal of the parotid gland revealed the duct lying underneath, unattached to the gland (*fig. 6h*), extending from a normal opening in the vestibulum oris to a deep-seated insertion confluent with the fibers of the temporal muscle, anterior to the ear. The explanation of this abnormality of gland and duct is suggested by the following account of the origin of the parotid gland (4).

The parotid is the first of the salivary glands to make its appearance, and "has been detected in an 8 mm. embryo as a furrow in the floor of the alveolobuccal groove in the neighborhood of the angle of the mouth." Initially small, "the furrow gradually elongates, and before the embryo has reached a length of 17 mm., it separates from the epithelium of the alveolobuccal groove and opening into the mouth cavity at a point which corresponds with the anterior end of the original furrow. Mesenchymatous tissue gradually forces its way between the tube and the alveolobuccal epithelium, and the tube, increasing in length, pushes its way back over the masseter muscle to the neighborhood of the external ear. As it comes into this region, the tube or duct as it may be called, begins to branch at its posterior extremity, the branches being at first solid outgrowths from the wall of the duct, and as these increase in number and size, and become surrounded by a mesenchymatous capsule, the gland assumes the position and general form of the adult structure. The accessory parotid gland arises as an outgrowth of the duct as it passes over the masseter muscle." Chievitz detected a branch arising from the duct at the anterior border of the masseter muscle in embryos of 12 weeks. He noted this branch passed deeply to come "into relation with the internal pterygoid muscle." The same structure was noted in embryos of 10 weeks, but in "this instance it had lost its connection with the parotid duct."

In the present instance the gland either became pinched off from the developing duct (possibly representing the accessory parotid gland because of its position on the masseter muscle), or corresponds to the detached portion observed by Chievitz. However, should this latter hypothesis be correct, the gland structure in this instance did not run in relation to the internal pterygoid muscle. The constricting fibers surrounding the gland probably correspond to the mesenchymatous capsule. Its attachment to the root of the nose and the extension of the parotid duct posteriorly suggests growth lacking some factor of control.

Masseter muscle. The anterior fibers of the superficial head of the masseter muscle extended from a position in line with the lower border of the malar bone corresponding to the lateral third of the orbit, and passed to

a normal insertion into the outer surface of the ramus of the mandible (*fig. 6, i*). The deep fibers occupied a normal position but were scanty.

Temporal muscle. The temporal muscle had a most unusual arrangement (*fig. 6, g*). It consisted of two layers. The superficial one filled the temporal fossa and inserted into the upper margin of the zygomatic process, thus becoming confluent by its attachment with the origin of those fibers of the masseter muscle that arose from the lower border of the zygomatic arch. The deep layer was sparse, attached itself to the deep fibers of the masseter, and ran in a normal manner under the zygomatic arch to attach to the coronoid process of the mandible.

Maxilla and mandible. Examination of maxilla and mandible proved them to have a normal amount of calcification, and the full complement of deciduous teeth. In the maxilla the incisor foramen was occluded, and search failed to reveal the naso-palatine or anterior palatine branches of the maxillary division of the n. trigeminus, although the palatine foramen was large. The roof of the mouth had a flat arch. Three foramena were found in the infra-orbital region, one in a normal position (marking the termination of the infra-orbital branch of the nerve facialis mentioned previously), the other two, medial to the former, and lying in close proximity to the nose, one placed above the other. The latter two foramena could easily be traced and joined about one-quarter inch posterior to their openings, each carrying a good-sized portion of the infra-orbital branch of the maxillary nerve. The lateral foramen prevented the introduction of a horse hair pathfinder, thus precluding identification. It is possible that this opening represents the small zygomatic foramen mesially misplaced.

Ernst Huber (5), speaking about the arrangement of the trigeminal foramena, says,

"the locations of these foramena in the lower primates appears to be determined by the groups of facial tactile vibrissae which receive their innervation through the corresponding cutaneous branches of the n. trigeminus. Examination of a large series of skulls of representatives of the various groups of the primates revealed the fact that the established ground plan of these foramena is retained throughout the ascending scale of primates. The variability in size and arrangement of the trigeminal foramena is considerable, and increase in their number is of common occurrence. In certain genera, this multiplicity of foramena appears to be the rule rather than the exception. Thus in higher platyrrhines and in lower catarrhines, the foramen infra-orbital may be replaced by three, four, five small foramena usually grouped in a cluster in the place where we should expect a single foramen. However, these small foramena may be arranged in a row along the infra-orbital margin as in certain representatives of the baboons. In others, especially in long-snouted baboons, the group of smaller infra-orbital foramena

come to be scattered over the upper surface of the maxilla, evidently because of the extreme growth of the maxilla. In the gibbons and in the anthropoid apes, the infra-orbital foramena tend to be reduced to a single foramen. In man this seems to be a rule, although double foramena are not unusual."

Enamel caps. Gross examination of the deciduous tooth germs disclosed enamel caps which easily could be removed from the underlying dentine and pulp, the cap maintaining its shape and having the consistency of thin celluloid. The enamel caps of the central and lateral incisors had a brownish appearance, dark at the tips, and graded to a lighter hue at the necks. The cuspids and molars, with the exception of the maxillary second molar, had this color confined to the tips of the cusps. The latter enamel cap presented none of this pigmentation. It is interesting to note that this coloring existed wherever the greatest amount of calcification normally would be anticipated. The specimen having been placed in formalin at the time of delivery and retained in this fixative during the progress of this investigation, oxidation does not present a likely explanation of this pigmentation. That it indicated the presence of blood is improbable, as "calcification does not occur readily where the blood supply is abundant" (6). The writer is grateful to Dr. Samuel W. Chase who offered the following information based on his unpublished findings:

"I have been investigating the changes occurring in recently formed enamel for the past two years. Among the changes or evidence of changes noted, has been this assumption of a brown color by the recently formed enamel in specimens preserved in formalin. What is behind the change I do not know, but in my specimens of human fetus, only the enamel which has been formed within three or four months becomes so discolored. Enamel formed longer than the three or four months does not become brown with formalin preservation. In the normal fetus, about the cervical two thirds of the enamel is browned, the occlusal third remaining white in the upper central incisor. In the upper lateral incisor all the enamel excepting a wedge-shaped portion in the middle of the incisal quarter is brown. All of the enamel of the cuspid and the two molars is brown. . . . To agree with my observations one would have to assume that your abnormal fetus had suffered considerable delay in enamelization, none of the enamel on the incisors, cuspids, or molars being more than three or four months old. The fact that brown shows only at the tips of the cuspids and molars may mean that only the tips have enameled. The absence of coloration of the upper second molar may mean absence of enamel. You state that the pigmentation of the incisors grades from dark to lighter passing from incisal to cervical. This I can interpret only as evidence that the enamel is thinner than in the normal term fetus and appears lighter cervically because it is thinnest there. I have noted this in the developing teeth of term pigs, where the enamel over the deciduous crowns is

thinner than that of the permanent teeth of pigs or that of the deciduous or permanent teeth of man.

"The brown enamel is strongly resistant to decalcification, whereas the enamel which remains white dissolves readily in decalcifying fluids, pointing to some chemical or physical difference in the two. What this difference is I have not been able to discover" (7).

Histologic data. Sections through the left mandible and molar tooth germs were prepared by the following technique. Following fixation in 10 percent formalin the tissue was placed in 2 percent HNO_3 for decalcification, washed for 6 hours in running water, placed in ascending alcohols and ether (each for 12 hours, then in thin celloidin (one week), and thick celloidin (one week), blocked, and 12μ sections cut, stained with alum hematoxylin and eosin, and mounted in gum damar.

Histologic examination disclosed that (a) odontoblasts and ameloblasts exhibited signs of degeneration; and (b) large calcospherites were present on the surface of the enamel (*figs. 7, 8*). There is an absence of calcium globuli in the dentine matrix, indicating poor calcification.

SUMMARY

Investigation of the head and neck of an anencephalic monster disclosed marked abnormalities which in some instances resembled an embryonic arrangement of tissue. This is readily understood when it is recognized that the function of the embryo is organogenesis and that in this instance arrest in development was due to absence of the influence of the central nervous system during fetal life (8). The quoted observations of Chase relative to pigmentation of the enamel caps suggest that the embryonic phase of enamelization was involved. The histologic evidence of poor calcification of the dentine as evidenced by a lack of globuli in the dentine matrix, and a tendency of the ameloblasts and odontoblasts to degenerate, further substantiates the opinion that the tooth buds exhibited an arrest in development.

The following abnormalities were encountered: Absence of brain, calvarium, and Gasserian ganglion; undifferentiated facial muscle group—quadratus labii superioris, zygomaticus major, and caninus; terminal branches of *n. facialis* distributed through facial muscle group instead of in its usual path through the parotid gland; embryonic arrangement of platysma fibers (sphincter colli); abnormal development of parotid gland and its duct; occluded incisor foramen; absence

of anterior palatine and naso-palatine nerves; arrest in development of the tooth buds. Additional interesting, but not abnormal findings, were a villus of mucous membrane in region of upper canine, and possibly a third infra-orbital foramena.

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MICROSCOPIC ANATOMY OF THE ENAMEL[1,2]

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Extraordinary difficulties are encountered in the study of human enamel because of the diminutiveness of its structural elements, its complexity, its variability from different angles of observation, its variability in different levels, and its confusing refractivity. Added to these obstacles are the bizarre patterns found which make it necessary to reconstruct the minutae of its histology from fragmentary evidence.

Between the years 1922 and 1932 I described, in seven publications (1, 2, 3, 4, 5, 6, 7), various details of enamel structure. With the help of models and photomicrographs the distribution of a system of interrodal capillary spaces, called tubes by early investigators, was traced. No attention was given to this work in the American literature. Whether this is due to a commendable conservatism on the part of anatomists, or to the inherent difficulties attending corroboration, or to an inexcusable inertia of those who are willing to build upon old fallacious theories instead of informing themselves concerning demonstrable facts (however difficult these may be) the use of radically erroneous theories continues to the detriment of progress.

The relation of the capillary space system, mentioned in my papers, to the striae of Retzius is one that has assumed importance in recent mottled enamel investigation. Theories explaining the formation of the Retzius' striae vary greatly. Hopewell-Smith (8), Noyes (9), Williams (10) and others regard pigment deposition as the principal cause of these markings. Others, including the great majority of modern students, have accepted the views of Salter (11) and John Tomes (12) that the Retzius' lines represent rhythmical pauses in

[1] Work supported in part by a Grant from the Research Commission of the American Dental Association.

[2] Read at the XIV Annual Meeting of the International Association for Dental Research, March 4th, 1936, Louisville, Ky.

enamel incrementation, causing well calcified layers formed during periods of maximum activity to be visually separated by lines of poorly calcified tissue formed in the intervals. This theory has recently been supported by Schour (13) who compares the process and appearance with the annular rings formed during tree growth.

The theory of rhythmicality in enamel deposition is based on unsupported conjecture. It is incredible that so many well known histologists have adopted it as a premise for teachings which must fall with their fundament.

Another school of students, including von Ebner (14), Czermak (15) and Caush (16) contend that the presence of rows of tubes situated between the prisms are the cause of the Retzius' striae.

An attempt to describe the anatomical distribution or morphology of this space system as a unit has never before succeeded, in fact, to the best of my knowledge, it has never been attempted.

Three fundamentally new anatomical features have thus far been presented in my work on this space system:

1. The inner regions of the enamel are divided into vertically arranged paralleled compartments by rows of interrodal capillary spaces (previously referred to as the parallel communications between the dento-enamel junction and the enamel's periphery (17)) (*figs. 1, 2, 3*).

2. The interrodal spaces, after peculiar changes in course, extend to the enamel's periphery, where they terminate in the valleys between the perikymata (Preiswerk) or imbrication lines (Pickerill) *figs. 4, 5, 6 and 7*. Experiments have shown that these spaces will conduct stain (or fluids) during the early post-eruptive life of the tooth.

3. Tufts were shown to be the cross sections of the vertically arranged rows of capillary spaces. It was furthermore shown that tufts were neither incompletely calcified rods nor artifacts.

To illustrate my findings plaster models showing incremental deposits, representing the supposed manner of enamel growth, were constructed (*fig. 8*). The imaginary lines of growth were duplicated by alternately dipping the model in thinly mixed plaster of paris darkened with lamp black and mixtures of pure plaster. The lateral incrementations were added with the help of a small brush. Sections of such a model present diagrammatically the lines of the theoretical

Fig. 1

Fig 2

Fig. 3

Fig. 4

Fig. 1. Paralleled rows of interrodal spaces, as seen in labial enamel of incisor

Fig. 2. 16 mm. view of rows sectioned near their point of origin.

Fig. 3. Continuations of rows seen in *Fig. 2.*

Fig. 4. Horizontal stripes in peripheral part of enamel caused by normal sinuosities of interrodal figures.

Fig. 5. Double curvatures of interrodal figures producing the horizontal stripes shown in *Fig. 4*.

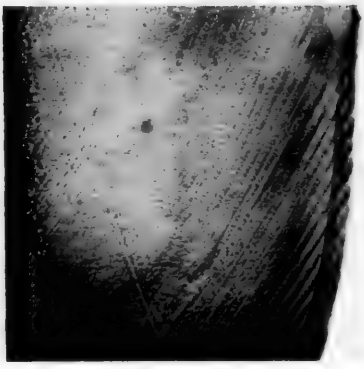

Fig. 6

Fig. 7

Fig. 6. Striae of Retzius continuous with valleys between enamel imbrications.
Fig. 7. Rows of tubes at edge of section leading into valleys shown in *Fig. 6*.

Fig. 8

Fig. 9

Fig. 10

Fig. 11

Fig. 12

Fig. 13

Fig. 8. Plaster model. See text.

Fig. 9. Longitudinal section of model shown in *Fig. 8.*

Fig. 10. Cross section of similar model.

Fig. 11. Diagonal cross section of similar model.

Fig. 12. Longitudinal section through enamel portion of model.

Fig. 13. Section of model showing labial hypoplasia extending through several layers of enamel.

incremental deposits. Each line on this model may be supposed to represent many of the stratifications found on the tooth.

Crown cross-sections and longitudinal sections made from the model apparently indicate, admittedly, the arrangement of the striae of

Retzius seen in our preparations (*figs. 9, 10, and 11*). However, if the labial enamel portion of the model is sectioned in a manner corresponding to our methods of making enamel rod cross sections from

Fig. 14. Theoretical "layers" of enamel, either of which, if existent, should be (theoretically) affected throughout by exanthematous fevers occurring during its formation.

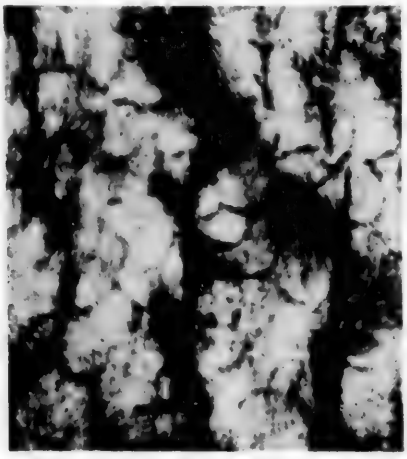

Fig. 15. Background shows frontal view of dento-enamel junction.

this region, the results obtained with the model fail to conform with anything found in the tooth section. The model exhibits a series of concentric rings (*fig. 12*) while in the tooth, the horizontally arranged

rows, in the superficial enamel regions, are superimposed on the vertically arranged basal part of the row in the deeper regions (*figs. 4 and 7*). Incremental lines are absolutely wanting.

The fallaceous belief of incremental enamel deposit is further controverted by the theory of pre-eruptive hypoplasia formation. The contention has been accepted by the profession that an exanthematous fever, occurring during the early stages of enamel deposition, will affect exactly those layers being laid down. If this assumption were correct, the result would not be a localized lesion extending through a large number of layers laid down at different times (*fig. 13*) but would involve the whole tissue of the enamel cap then in process of construction (*fig. 14*). The same principle would apply also to the mechanism of mottled enamel production.

The mosaic pattern exhibited by a frontal view of the dento-enamel junction is exhibited by exposure through the paralleled rows. The rings in the background are caused by the papilliform projections of the dention into the enamel (*fig. 15*).

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A QUANTITATIVE ROENTGENO-DENSITOMETRIC STUDY OF THE CHANGES IN TEETH DUE TO ATTRITION[1, 2]

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Probably the first observations of change in dentine exposed by attrition of the occlusal surfaces of teeth were limited to its polished and often stained appearance. The exposed dentine not only was free from dental caries but actually seemed harder, clinically, to the point of the explorer than ordinary dentine, a fact which has been variously attributed (a) to a packing effect by the forces of mastication, (b) to a chemical change due to the action of the saliva or (c) to a physiological building up of a defense by the tooth.

When a labio-lingual section is cut through the center of an abraded tooth, reflected light usually shows a symmetrical, white cone (1) with its base at the occlusal surface and its apex toward the pulp chamber. This cone-shaped changed area is opaque to transmitted light and has been called opaque sclerosed dentine (1). Between the apex of this cone and the existing pulp chamber, another cone is frequently discovered with its apex at the apex of the "attrition" cone and its base at the surface of the pulp chamber. This latter cone of secondary dentine which appears dark by reflected light but transparent by transmitted light is called "transparent" dentine. By the proper staining technic (1), (2) these cones may be demonstrated to be impermeable to stain whereas the remaining dentine, if free from other change (such as accompanies caries, age, etc.), may be heavily

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[2] Awarded the Morris L. Chaim Prize by the First District Society of the State of New York; read at the New York State Dental Centennial Meeting, December 7, 1934.

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impregnated with stain. The impermeability has been variously attributed to a degenerative change of the contents of the dentinal tubules (12) perhaps involving a fatty degeneration or occlusion (3), to an occlusion of the tubule openings due to the sealing effect of secondary dentine with diminution of the lumen of the tubules and an obliteration of the tubule projections into the matrix (10). With respect to the attrition cone of opaque sclerosed dentine, a certain credence for the last named explanation is found in the appearance of roentgenograms of these sections (5). Corresponding with the attrition cone of the section there is a light area in the film image of the dentine. Corresponding with the secondary dentine cone there is an area darker than the image of the attrition cone and in most instances even darker than the unchanged dentin of the bulk of the tooth. Comparing the attrition cone and the transparent dentine cone (1), both are chromophobic, (2) the former is light opaque and more radiopaque, the latter light translucent and less radiopaque. The lesser radiopacity of the secondary dentine cone substantiates the hypothesis of Boedecker (3), Fish (2), Kimmelstiel (12) and others who state that the translucency is due to the presence of organic changes, possible fatty degeneration. The greater radiopacity of the attrition cone may be attributed to calcific deposition as suggested previously by Beust (10) and others. No one, however, has been able to describe these changes quantitatively. Some new data, using a densitometric study of roentgenograms (6), by which the changes occurring in dentine exposed by attrition are measured is presented herewith.

The precise measurement of the roentgen-ray absorption of dentine (*fig. 1*) is made possible by the method described by Warren et al. (6). The method cannot be used to estimate the magnitude of the "line" changes observable in many roentgen-ray pictures of tooth slabs. [For example, the highly opaque line in the dentine under occlusal caries shown in Tooth No. 3, Van Huysen et al. (7).] However, this method does give a number representing the roentgen-ray absorption of a given area in the dentine, it is well suited to the task of determining the roentgen-ray absorption of "normal" dentine and it is applicable to the measurement of changes in dentine where relatively large areas are involved. Fortunately, the areas of changed dentine

under attrition are sufficiently large in most cases to permit study by this method.

Using the densitometric method for measuring the roentgen-ray absorption of 13 anterior teeth showing abraded incisal surfaces free of enamel, and 1 canine with intact enamel, it is proposed in each

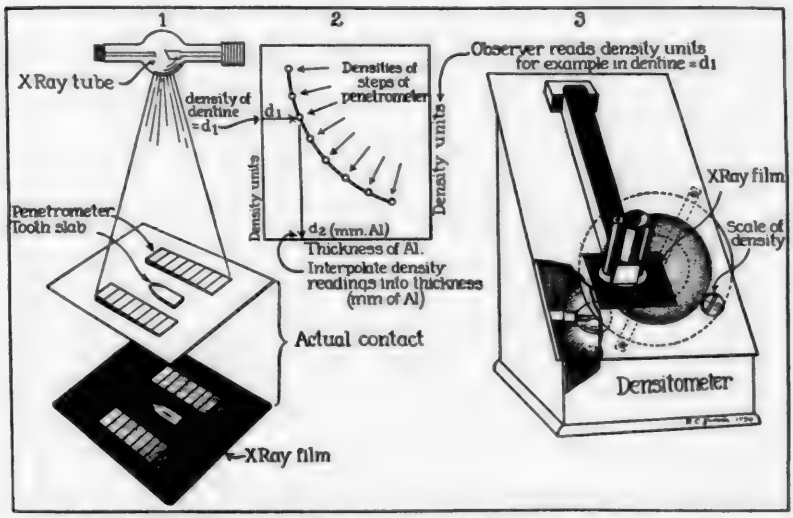

Fig. 1. Schematic drawing of precision densitometric method for measuring roentgen-ray absorption of dentine. Under 1, an x-ray tube exposes a film upon which aluminum penetrometers and tooth slabs are placed. The developed film is represented at lower left. Under 3, a Capstaff-Purdy densitometer is illustrated which reads densities of various steps of penetrometer image in developed x-ray film. The average densities in the various steps are then plotted as ordinates against respective thicknesses of these steps in mm. (under 2). Then a density reading in the dentine (for example, d_1) is located on the ordinate, followed across horizontally until curve is reached and by dropping perpendicular to base line, exact thickness of Al which would cast same darkness of shadow on x-ray film is found. This thickness (mm. Al) is called *Interpolated Value* and may be reproduced within 5 percent by repeated exposures, plottings, and interpolations.

tooth (a) to establish an average absorption value for the visibly unchanged "normal" dentine, (b) to measure the change in the roentgen-ray absorbing ability in the "attrition" cones, (c) to measure the absorption in the secondary dentine and compare it to the normal roentgen-ray absorption value and (d) to measure the roentgen-ray absorption in the dentine in the root portion of the tooth.

PROCEDURE

A complete description of every phase of the method has been previously published (6). From *fig. 1* the general nature of the procedures may be understood.

In order that the triplicate interpolated values given in the various tables later may be clearly evaluated, the actual interpolation procedure is included using an area in Tooth No. 21 as an example. Tooth No. 21 is roentgen-rayed in triplicate on films #10, #11, and #12 at the standardized exposure of 30 KV, 30 ma, 15 inches, ¼ seconds, no tube stand filter, and using black-paper-wrapped films. Densitometer readings in the various

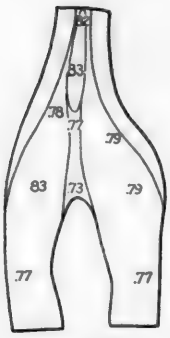

Fig. 2

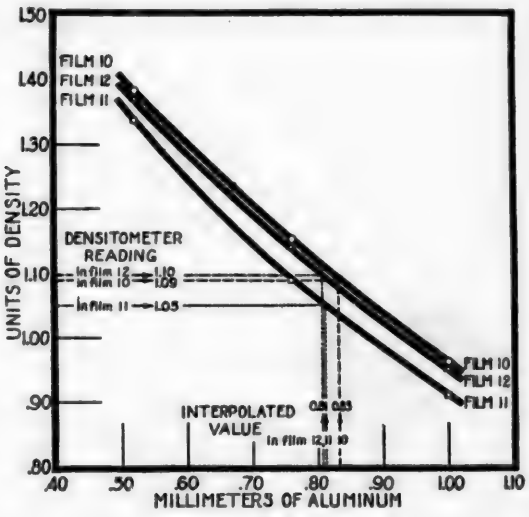

Fig. 3

Fig. 2. Outline Drawing of Tooth No. 21 showing carefully selected areas in which densitometer readings are made. Numbers appearing in outline drawing are average interpolated values (see Table II).

Fig. 3. The actual interpolation of densitometer readings in attrition cone of Tooth No. 21. Densitometer readings (along ordinate at right hand side of graph) are interpolated (follow broken lines) into interpolated values for attrition cone. Note that interpolated value from Films 11 and 12 is the same number, i.e., 0.81 mm. Al.

steps of the penetrometers in each film are averaged and the averages (Table I) plotted as ordinates to obtain typical density-thickness curves (for example, *fig. 1*—under 2). Then densitometer readings are made in each film in carefully selected areas in the tooth slab image (*fig. 2*) and recorded in Table II under *densitometer readings*.

To illustrate the actual interpolation the small section of each of the three density-thickness curves from Films 10, 11, and 12 including the densitometer readings in steps 2, 3, and 4 are shown in *fig. 3*. The densitometer reading (1.09) in the attrition cone in Film 10 is observed to cut the density-thickness curve at a value of 0.83 mm. Al; this is

the interpolated value for Film #10. Similarly the densitometer readings in the same area in Films #11, #12 (1.05 and 1.10 respectively) are observed to cut their respective density-thickness curves at a value of about 0.81 mm. Al; this is the interpolated value for Films #11 and #12. (Compare Table II under interpolated value). It is noted

TABLE I

Average densitometer readings in the various steps of the penetrometers to be plotted against the thickness of step (see Fig. 3)

ALUMINUM PENETROMETER		AVERAGE DENSITOMETER READINGS		
Step	Thickness	Film 10	Film 11	Film 12
	(mm.)			
1	0.26	1.77	1.72	1.76
2	0.52	1.39	1.34	1.38
3	0.76	1.15	1.09	1.14
4	1.00	0.96	0.91	0.95
5	1.24	0.83	0.77	0.82
6	1.48	0.72	0.68	0.70
7	1.76	0.62	0.59	0.60
8	2.01	0.57	0.55	0.56

TABLE II

Complete data for tooth no. 21

	DENSITOMETER READINGS			INTERPOLATED VALUES			AVERAGE INTERPOLATED VALUES	
	Film 10	Film 11	Film 12	Film 10	Film 11	Film 12		
A. Attrition cone								
Incisal	1.09	1.05	1.10	0.83	0.81	0.81	0.82	0.82
Pulpal	1.10	1.02	1.09	0.82	0.84	0.82	0.83	
B. Secondary dentine								
Occlusal	1.13	1.07	1.14	0.78	0.78	0.76	0.77	0.75
Pulpal	1.17	1.13	1.17	0.73	0.73	0.73	0.73	
C. Normal coronal dentine								
Middle third	1.15	1.06	1.12	0.76	0.79	0.78	0.78	0.80
	1.14	1.03	1.14	0.77	0.83	0.76	0.79	
Gingival third	1.09	1.04	1.08	0.83	0.82	0.83	0.83	
	1.09	1.08	1.13	0.83	0.77	0.77	0.79	
D. Root dentine	1.13	1.08	1.12	0.78	0.77	0.78	0.78	0.77
	1.15	1.08	1.13	0.76	0.77	0.77	0.77	

that two or more films may have exactly the same interpolated value for the same area in the dentine. The average interpolated value of 0.82 mm. Al is easily calculated. By exactly identical procedure, the densitometer readings in the other areas (Table II) are recorded, interpolated, and averaged. For the sake of brevity only the interpolated values for the various areas in the other teeth are tabulated herewith.

DISCUSSION

During mastication, teeth whose occlusal surfaces come in contact with each other undergo a certain amount of attrition of these occluding surfaces. The degree of wear of the surfaces is determined primarily by the use to which the individual's teeth have been put. The three main factors concerned are: (1) the manner in which these teeth come in contact, i.e., the greatest amount of attrition occurs in those surfaces that have the greatest amount of contact; (2) the amount of foreign matter of gritty substance, principally sand, contained in the materials taken into the mouth; and (3) the length of time over which teeth have been used for chewing, i.e., the individual's tooth age. Other factors are the muscular development of the jaws (which may be a function of heredity and development) and probably to a certain extent the structure of the teeth, (although it is not known at the present time how much actual difference there may be in the structure of teeth in the same mouth or from individual to individual). The most important factors, therefore, seem to be the type of occlusion, the type of materials introduced into the mouth, and the tooth age of the individual. For the teeth reported in this paper, the occlusion and type of food as well as the development of the individual's masticatory muscles is not known.

The only known factor relative to the amount of attrition of the teeth in this study is the age of the individual. Table No. 17 giving the summary of the data shows the number of teeth studied and the age of the individuals from whom these teeth were extracted. Teeth #7, #19, #20, #21, #22, #23, and #26 were extracted from a male of 41 years of age; teeth #49, #51, #52, #53, #54, and #55 were extracted from a female 47 years of age; and #39, the non-attrited canine, from a female 15 years of age. It has been shown that dentine differs in its permeability to stains (1) and roentgen-ray absorption as the age of the individual is increased (13). Therefore one tooth with relatively unchanged dentine from a young 15 year old female is included in the study for comparative purposes. All the worn teeth may be described as showing typical amounts of attrition for middle aged people.

In our discussion we are suggesting a grouping of the teeth based on the roentgen-ray absorption as seen in the roentgenogram and

supplemented by the appearance of the tooth slab. In the young tooth with intact enamel, the dentine is homogeneous in its roentgen-ray absorption (*fig. 4*) as well as in appearance (*fig. 6*) having no streaks or areas of opaque or transparent dentine. In the worn teeth, the dentine presents certain areas of changed roentgen-ray absorption as well as appearance; sometimes there is a relatively large attrition cone of radiopaque and light opaque dentine (*fig. 4*) with only a tiny dot of transparent secondary dentine (*fig. 6*); sometimes the attrition cone and the secondary dentine cone are almost equal in size (*fig. 8*) presenting a typical "hour glass" appearance (*fig. 4*) with the attrition cone more radiopaque and the secondary dentine more radiolucent than normal dentine in the same tooth; and finally sometimes there is a relatively large cone of transparent, secondary dentine (*fig. 10*) with only a tiny, opaque attrition cone (*fig. 4*) without marked changes in the roentgen-ray absorption of these cones.

Tooth No. 39 is a typical young tooth with intact enamel (from a 15 year old female) and clearly shows (*fig. 4*, No. 39) the homogeneous appearance of the dentine. Teeth Nos. 49, 52, 53, and 55 show a large attrition cone (No. 53 for example in *fig. 4*) with only a tiny dot of transparent dentine. Teeth Nos. 7, 20, 21, 22, and 26 show attrition cones and secondary dentine cones of about the same size. (For example, No. 21 in *fig. 4*). Teeth Nos. 19, 23, 51, and 54 show a relatively large cone of secondary dentine with a very small attrition cone. (For example, *fig. 4*, No. 51.)

For clarity in the discussion of the data, four areas in each tooth section are considered: (A) the attrition cone, (B) the secondary dentine, (C) the normal coronal dentine, and (D) the root dentine.

Normal, Young Tooth: Tooth No. 39: ***fig. 4***, No. 39 shows a half tone drawing of the roentgenogram of an 0.84 mm. thick section of a lower left canine extracted from a 15 year old female. The enamel covering the crown is intact and the dentine appears of a uniform density throughout the tooth. The picture made by transmitted light, *fig. 6*, shows a uniform opacity of the greater part of the dentine in the center of the section and well up into the incisal tip. There is a wide margin of translucent dentine just inside of the dentino-enamel junction and some visible modification (10) of the dentine at the root end of the section more on the right than at the left. *Fig. 5* shows an outline drawing of this section with the interpolated values (Table III) in the areas measured.

In the coronal dentine, the averaged interpolated values are: 0.82, 0.82, 0.81, and 0.81 mm. Al showing a high degree of constancy of absorption. In the root dentine, the average interpolated values are 0.76, 0.76, 0.75, and 0.76 mm. Al again showing a

high degree of constancy. It is apparent that the coronal dentine is about 7 percent more radiopaque than the root dentine.

Inasmuch as the areas of changed dentine which are apparent under attrition are all located in the coronal dentine, the interpolated values of this portion of the normal, young tooth are particularly of interest. The absorption values of this coronal dentine are described as normal not so much because they represent a "normal" absorption for a given thickness of dentine but because of their unusual constancy they can be taken as evidence of a homogeneous, "primitive, unmodified dentine" (10).

TABLE III

Tooth no. 39: Normal young tooth with intact enamel and homogeneous dentine

	INTERPOLATED VALUES			
	Film 22	Film 23	Film 24	Average
C. Normal coronal dentine				
Occlusal third	0.82	0.83	0.81	0.82
Middle third	0.80	0.82	0.80	0.81
	0.79	0.84	0.82	0.82
	0.81	0.80	0.81	0.81
D. Root dentine				
Gingival	0.76	0.75	0.78	0.76
	0.76	0.77	0.76	0.76
Apical	0.74	0.75	0.75	0.75
	0.76	0.77	0.76	0.76

In the teeth showing attrition, it is impossible to determine whether or not the dentine outside of the changed areas, i.e., outside the attrition cone and the secondary dentine cone, is different than the dentine of the tooth would have been without the effects of attrition. However, because its roentgen-ray absorption is constant over relatively large areas in the crown and because it is homogeneous in appearance, we are calling it normal coronal dentine. We are comparing the roentgen-ray absorption of this homogeneous dentine with the absorption of the changed areas in the same tooth.

Prominent Attrition Cone: Tooth No. 53: A lower canine, was extracted from a woman aged 47 years. It shows (*fig. 6*) loss of the enamel over the incisal tip and slight loss of underlying dentine due to attrition. *Fig. 4* is a half tone drawing of the roentgen-ray image of the 0.84 mm. thick section. This illustration shows a well outlined pulp

chamber and uniform density of the dentine excepting the cone-shaped area with its base at the incisal edge and the apex at the tip of the pulp chamber. *Fig. 6*, a transmitted light photograph, shows a tiny bit of transparent dentine at the point of the existing pulp chamber; the highly opaque area just beneath the transparent dot is an accidental stain in the pulp chamber from an unknown source (perhaps blood) and is not solid tooth substance. The attrition cone is clearly indicated as a narrow band of opaque dentine (9). The average interpolated values in the attrition cone (*fig. 5*) are 0.84 and 0.86 mm. Al (Table IV), whereas in the normal coronal dentine, whose homogeneous appearance may be noted from the half-tone of the roentgen-ray, the interpolated values are 0.78 and 0.79 mm. Al. This 10 percent increase in opacity may be attributed to a deposition of calific material (10) and cannot possibly be interpreted as due only to soft tissue necrosis or other changes. Unfortunately, this method of study will not permit a differentiation between a pathological "mechanical precipitation" and a

TABLE IV

Tooth no. 53: Prominent attrition cone, tiny transparent secondary dentine cone

	INTERPOLATED VALUES			
	Film 29	Film 30	Average	
A. Attrition cone				
Incisal	0.83	0.85	0.84	
Pulpal	0.85	0.86	0.86	
B. Secondary dentine				
Pulpal	0.80	0.80	0.80	
C. Normal coronal dentine				
Middle third	0.78	0.77	0.78	0.78
	0.80	0.78	0.79	
D. Root dentine	0.78	0.76	0.77	0.79
	0.80	0.79	0.80	

physiological deposition of solids in the dentine. The amount of filling in of any one tubule is indeterminable by this method, only gross changes involving the tubules in a comparatively large area being measured. The average interpolated value in the tiny area of secondary dentine is 0.80 mm. Al; a value that, although it is 2½ percent greater than the normal coronal dentine, cannot be considered as being different from the normal. In the root, average interpolated values of 0.77 and 0.80 mm. Al are found, showing little or no difference from values in the crown.

Tooth No. 52: A lower right incisor from a 47 year old female, presents a typical picture of attrition with a tiny area of secondary dentine, (*fig. 6*) between a relatively large attrition cone and the tip of the pulp chamber. From an 0.85 mm. thick section (*fig. 5*) average interpolated values of 0.85 and 0.84 mm. Al are obtained in the attrition cone which show an increase of 7½ percent compared to the absorption of normal coronal dentine in which average interpolated values of 0.80, 0.77, 0.79, and 0.79 mm. Al are found (Table V). The average interpolated value of 0.79 mm. Al in the root dentine shows no evidence of difference between crown and root.

Tooth No. 55: A lower right incisor from a 47 year old female, presents a picture of typical abrasion in which there is a very small area of secondary dentine between a large attrition cone and the tip of the pulp chamber (*fig. 6*). In the outline drawing (*fig. 5*) of the roentgenogram of the 0.83 mm. thick section, the average interpolated

TABLE V

Tooth no. 52: Prominent attrition cone, tiny transparent secondary dentine cone

	INTERPOLATED VALUES		
	Film 29	Film 30	Average
A. Attrition cone			
Incisal	0.87	0.83	0.85
Pulpal	0.85	0.82	0.84
B. Secondary dentine			
C. Normal coronal dentine			
Middle third	0.80	0.79	0.80
	0.78	0.76	0.77
	0.81	0.76	0.79
	0.81	0.77	0.79
D. Root dentine	0.81	0.76	0.79

TABLE VI

Tooth no. 55: Prominent attrition cone, tiny transparent secondary dentine cone

	INTERPOLATED VALUES		
	Film 29	Film 30	Average
A. Attrition cone			
Incisal	0.81	0.82	0.82
Pulpal	0.81	0.82	0.82
B. Secondary dentine			
C. Normal coronal dentine			
Middle third	0.78	0.76	0.77
Gingival third	0.77	0.74	0.76
D. Root dentine			
Gingival	0.77	0.75	0.76
Apical	0.76	0.73	0.75

values of 0.82 and 0.82 mm. Al in the attrition cone (Table VI) show an increase of 7 percent over the average interpolated values of 0.77 and 0.76 mm. Al in the normal coronal dentine. The average interpolated values of 0.76 and 0.75 mm. Al in the root dentine are not significantly different from those in the crown.

Tooth No. 49: The 0.84 mm. thick section of a lower canine from a 47 year old female, shows (*fig. 6*) a small area of transparent secondary dentine and a relatively large opaque

attrition cone. The average interpolated values in the attrition cone (*fig. 5*) are 0.83 and 0.82 mm. Al (Table VII) showing an increase of 5 percent over the average interpolated values of 0.81, 0.80, 0.78, and 0.80 mm. Al in the normal coronal dentine. The average interpolated value of 0.79 mm. Al in the secondary dentine shows no difference between its roentgen-ray absorbing ability and that of normal dentine. There is also no real difference between normal crown and root dentine, the latter showing average interpolated values of 0.77 and 0.78 mm. Al.

In summary, in the four teeth showing a prominent attrition cone and a tiny bit of transparent secondary dentine, the attrition cone showed increases of radiopacity of 10, 7½, 7, and 5 per cent over normal

TABLE VII

Tooth no. 49: Prominent attrition cone, tiny transparent secondary dentine cone

	INTERPOLATED VALUES				
	Film 25	Film 26	Film 27	Average	
A. Attrition cone					
Incisal	0.82	0.85	0.81	0.83	
Pulpal	0.79	0.87	0.79	0.82	
B. Secondary dentine					
Pulpal	0.76	0.83	0.77	0.79	
C. Normal coronal dentine					
Occlusal third	0.80	0.83	0.79	0.81	0.79
	0.79	0.83	0.79	0.80	
Middle third	0.75	0.83	0.77	0.78	
	0.77	0.84	0.78	0.80	
D. Root dentine	0.76	0.78	0.76	0.77	
	0.77	0.80	0.78	0.78	

coronal dentine. This increase in radiopacity offers no evidence as to the cause of the change. However, the radiopaque attrition cone penetrates too deeply into the dentine to be plausibly attributed to a packing effect of mastication. We cannot tell whether radiopacity results from a chemical action of the saliva or from "physiological changes in the dentine" (1). Secondary dentine and root dentine could not clearly be shown to be different from crown dentine in radiopacity.

Equal Sized Attrition Cone and Transparent Secondary Dentine Cone: Tooth No. 21: Fig. 8, a lower right incisor, was extracted from a 41 year old man. It shows complete loss of the enamel over the incisal tip and slight attrition of the underlying dentine.

Fig. 4, a half tone drawing of the roentgen-ray image of the 0.85 mm. section taken from it, shows a thin elongated oval of increased density in the center of the incisal half of the dentine. The oval shape of the opaque dentine may be explained by considering that any section through a cone that does not pass through the base results in an oval, or it may be explained on anatomical grounds. Extending from the attrition cone to the pulp chamber and embracing the tip of the attrition cone and the tip of the pulp chamber there is a cone of transparent secondary dentine of decreased density. The two cones apex to apex are shaped somewhat like an hour glass. The remainder of the dentine of the crown and root appears to be of a uniform density. The outline drawing (*fig. 7*) shows that this major part of the normal coronal dentine gives average interpolated values of 0.78, 0.79, 0.83, and 0.79 mm. Al. Normal dentine for this tooth therefore has an average value of 0.80 mm. Al (Table VIII). The increased density

TABLE VIII

Tooth no. 21: Equal sized attrition and transparent secondary dentine cones

	Interpolated values				
	Film 10	Film 11	Film 12	Average	
A. Attrition cone					
Incisal	0.83	0.81	0.81	0.82	
Pulpal	0.82	0.84	0.82	0.83	
B. Secondary dentine					
Occlusal	0.78	0.78	0.76	0.77	0.75
Pulpal	0.73	0.73	0.73	0.73	
C. Normal coronal dentine					
Middle third	0.76	0.79	0.78	0.78	0.80
	0.77	0.83	0.76	0.79	
Gingival third	0.83	0.82	0.83	0.83	
	0.83	0.77	0.77	0.79	
D. Root dentine	0.78	0.77	0.78	0.78	
	0.76	0.77	0.77	0.77	

shown in the elongated oval in the incisal half of the dentine measures 0.82 and 0.83 mm. Al showing that there is an actual increase of 4 percent in the dentine of the attrition cone, which is attributable only to an increase in the calcific material. Between the area of increased density and the pulp chamber in what appears to be transparent secondary dentine, (*fig. 8*), the average interpolated values of 0.73 mm. Al near the pulp chamber shows that part of the secondary dentine is 5 to 6 percent less dense than the normal dentine. However, a measurement of 0.77 mm. Al in the same cone of secondary dentine but occlusally at a point where the tip of the original pulp chamber was located indicates that the older part of this dentine has become about as dense as that of the normal coronal dentine.

In the root dentine, average interpolated values of 0.77 and 0.77 mm. Al are found which are about 4 percent less than the interpolated values found in the crown.

Tooth No. 7: An upper left canine from a 41 year old male, shows (*fig. 8*) an area of

transparent secondary dentine between the attrition cone and the tip of the pulp chamber. When interpolated values are obtained from a roentgenogram of an 0.86 mm. thick section, the attrition cone shows (*Fig. 7*, Table IX) average interpolated values of 0.82 and 0.83 mm. Al, an increase of about 7½ percent over the average interpolated values of 0.78, 0.78, 0.76, 0.77, 0.76, and 0.78 mm. Al found in the normal coronal dentine. The occlusal part of the secondary dentine shows an increase of about 4 percent over the normal dentine but the pulpal portion is of about the same opacity as the normal dentine.

Tooth No. 20: A lower right incisor from a 41 year old male, shows (*fig. 8*) a well defined triangular area of secondary dentine between an opaque attrition oval and the pulp chamber. The interpolated values are found from the roentgenogram of an 0.85 mm. thick section. The average interpolated value (*fig. 7*, Table X) in the attrition cone

TABLE IX

Tooth no. 7: Equal sized attrition and transparent secondary dentine cones

	Interpolated values				
	Film 4	Film 5	Film 6	Average	
A. Attrition cone					
Incisal	0.86	0.82	0.77	0.82	0.83
Pulpal	0.86	0.82	0.80	0.83	
B. Secondary dentine					
Occlusal	0.83	0.80	0.76	0.78	0.79
Pulpal	0.80	0.76	0.75	0.77	
C. Normal coronal dentine					
Occlusal third	0.82	0.77	0.76	0.78	0.77
	0.81	0.76	0.77	0.78	
Middle third	0.78	0.77	0.73	0.76	
	0.80	0.77	0.73	0.77	
Gingival third	0.80	0.76	0.73	0.76	
	0.80	0.77	0.76	0.78	
D. Root dentine					

is 0.83 mm. Al, a value about 4 percent greater than the normal coronal dentine whose average interpolated value is 0.80 mm. Al. Running down from the incisal edge a little way into the attrition cone in nearly the center of the dentine is a stained crack which appears much like a dark hair in the section and also in the roentgenogram. Its influence is easily seen in the low average interpolated value of 0.79 mm. Al which appears near the incisal edge (*fig. 7*). The average interpolated values in the secondary dentine, 0.76 mm. Al, shows that it is less radiopaque by about 5 per cent than the normal coronal dentine. The root dentine is also less radiopaque (by 6 percent) than the crown dentine.

Tooth No. 22: A lower left incisor from a 41 year old male, shows (*fig. 8*) a well defined cone of transparent secondary dentine between the attrition oval and the pulp chamber. From the roentgenogram of the 0.85 mm. thick section, the average interpolated value in the attrition oval (*fig. 7*, Table XI) is 0.81 mm. Al, an increased radiopacity of only 1 percent as compared to the normal coronal dentine whose average

interpolated value is 0.80 mm. Al. However, the secondary dentine shows a decreased absorption of 5 percent. The root dentine also is less radiopaque by 2½ percent than the crown dentine.

TABLE X

Tooth no. 20: Equal sized attrition and transparent secondary dentine cones

	INTERPOLATED VALUES				
	Film 10	Film 11	Film 12	Average	
A. Attrition cone					
Incisal	0.78	0.78	0.80	0.79	
Pulpal	0.83	0.82	0.83	0.83	
B. Secondary dentine					
Pulpal	0.79	0.74	0.74	0.76	
C. Normal coronal dentine					
Occlusal third	0.82	0.79	0.80	0.80	
	0.80	0.79	0.78	0.79	0.80
Gingival third	0.83	0.81	0.80	0.81	
	0.82	0.81	0.80	0.80	
D. Root dentine		0.76	0.73	0.75	
		0.76	0.73	0.75	

TABLE XI

Tooth no. 22: Equal sized attrition and transparent secondary dentine cones

	INTERPOLATED VALUES				
	Film 10	Film 11	Film 12	Average	
A. Attrition cone					
Incisal	0.83	0.77	0.83	0.81	
Pulpal	0.83	0.81	0.78	0.81	
B. Secondary dentine					
Pulpal	0.77	0.75	0.76	0.76	
C. Normal coronal dentine					
	0.80	0.82	0.77	0.80	
Gingival third	0.82	0.82	0.80	0.81	0.80
	0.80	0.76	0.80	0.79	
	0.80	0.79	0.77	0.79	
D. Root dentine		0.78	0.80	0.79	0.78
		0.76	0.78	0.77	

Tooth No. 26: A lower left incisor from a 41 year old male, shows (*fig. 8*) a fairly large area of transparent secondary dentine between the attrition oval and the pulp chamber. The roentgen-ray absorption measurements on an 0.85 mm. thick section

(*fig. 7*) indicate an average interpolated value of 0.82 mm. Al for the attrition cone which is only 2 percent greater than the normal coronal dentine which has an average interpolated value of 0.80 mm. Al (Table XII). As in Tooth No. 20, there is a line running down a minute distance from the incisal edge into the attrition cone. Its influence may be seen in the low average interpolated value of 0.75 mm. Al at the incisal edge (*fig. 7*). The secondary dentine, with average interpolated values of 0.75 and 0.72 mm. Al, shows a marked decrease of about 9 percent in radiopacity compared to normal coronal dentine. The root dentine also is less radiopaque (by 6 percent) than the crown dentine.

In summary, it has been shown that in 5 teeth in which the attrition cone and the transparent secondary dentine cone are both fairly

TABLE XII

Tooth no. 26: Equal sized attrition and transparent secondary dentine cones

	INTERPOLATED VALUES				
	Film 13	Film 14	Film 15	Average	
A. Attrition cone					
Incisal	0.76	0.77	0.73	0.75	
Pulpal	0.82	0.85	0.80	0.82	
B. Secondary dentine					
Occlusal	0.73	0.76		0.75	0.73
Pulpal	0.71	0.73		0.72	
C. Normal coronal dentine					
Middle third	0.79	0.79	0.78	0.79	0.80
	0.83	0.80	0.82	0.82	
D. Root dentine		0.75	0.75	0.75	
		0.75	0.76	0.75	

large and of approximately equal size, giving rise to an "hour glass" shape in the roentgenogram, that the attrition cone has an increased radiopacity of 4, 7½, 4, 1, and 2 percent; which can be attributed to an increase in calcific material in the affected tubules. The secondary dentine has a marked decrease in radiopacity of −6, +2½, −5, −5, and −9 percent, respectively; the root dentine also is of less radiopacity by 4, 0, 6, 2½ and 6 percent than normal coronal dentine.

Prominent Transparent Secondary Dentine Cone: Tooth No. 51: A lower left incisor from a 47 year old female, shows (*fig. 10*) a prominent cone of transparent secondary dentine between the moderately attrited, deeply grooved, incisal edge and the pulp chamber. In the half-tone (*fig. 4*) of the roentgenogram of the 0.84 mm. thick section, there is evident a broad cone of decreased density with its base at the flattened tip of the pulp chamber and its truncated tip just beneath the incisal edge. On each side of

the cone of decreased density a line of increased density is visible, a line so thin that measurement is out of the question. An almost insignificant spot of increased radio-

TABLE XIII

Tooth no. 51: Small attrition cone and prominent transparent secondary dentine cone

	INTERPOLATED VALUES			
	Film 25	Film 26	Film 27	Average
A. Attrition cone				
B. Secondary dentine				
Occlusal	0.73	0.79	0.70	0.74
Pulpal	0.74	0.78	0.70	0.74
C. Normal coronal dentine				
Occlusal third	0.73	0.79	0.73	0.75
	0.75	0.80	0.70	0.75
Middle third	0.74	0.80	0.73	0.76
	0.74	0.79	0.74	0.76
D. Root dentine	0.74	0.78	0.73	0.75
	0.74	0.79	0.74	0.76

TABLE XIV

Tooth no. 19: Small attrition cone and prominent transparent secondary dentine cone

	INTERPOLATED VALUES				
	Film 10	Film 11	Film 12	Average	
A. Attrition cone					
Incisal	0.88	0.82	0.86	0.85	
B. Secondary dentine					
Occlusal	0.83	0.79	0.84	0.82	
Middle	0.83	0.81	0.81	0.82	0.81
Pulpal	0.80	0.79	0.82	0.80	
C. Normal coronal dentine					
Occlusal third	0.83	0.83	0.81	0.82	
	0.82	0.78	0.81	0.80	
Middle third	0.88	0.82	0.83	0.84	0.82
Gingival third	0.82	0.78	0.82	0.81	
	0.87	0.82	0.82	0.83	
	0.82	0.79	0.83	0.81	
D. Root dentine		0.74	0.78	0.76	0.77
		0.76	0.79	0.77	

pacity just at the incisal edge gives an indication of what may have been the apex of the attrition cone. The average interpolated values (see *fig. 9*, Table XIII) in the transparent secondary dentine, i.e., 0.74 and 0.74 mm. Al, are about 2½ percent less than the

average interpolated value of 0.76 mm. Al in the normal coronal dentine. The root dentine is of the same radiopacity as the crown.

TABLE XV

Tooth no. 23: Small attrition and prominent transparent secondary dentine cone

	INTERPOLATED VALUES				
	Film 10	Film 11	Film 12	Average	
A. Attrition cone					
Incisal	0 89	0.84	0.83	0 85	
B. Secondary dentine					
Pulpal	0.77	0 76	0.81	0.78	
C. Normal coronal dentine					
Occlusal third	0.84	0.82	0 86	0 84	
	0.83	0.78	0.82	0.81	
Middle third	0.82	0 79	0.81	0.81	0.81
	0 78	0.75	0.77	0.77	
Gingival third	0.83	0.82	0 82	0.82	
	0.84	0.82	0 86	0.84	
D. Root dentine		0 79	0 79	0 79	
		0.78	0 81	0 80	

TABLE XVI

Tooth no. 54: Small attrition cone and prominent transparent secondary dentine cone

	INTERPOLATED VALUES			
	Film 29	Film 30	Average	
A. Attrition cone	0 85	0.87	0 86	
B. Secondary dentine	0.83	0 85	0 84	
C. Normal coronal dentine				
Occlusal third	0.81	0.82	0.82	
	0.81	0 79	0 80	0 81
Middle third	0 81	0 80	0 80	
	0.80	0.82	0 81	
D. Root dentine	0.78	0 77	0 78	
	0.78	0.77	0.78	

Tooth No. 19: A lower left canine from a 41 year old male, shows (*fig. 10*) a slightly attrited tip with a prolonged, slender cone of transparent secondary dentine with its base at the pulp chamber and its tip near the apex of the relatively small attrition cone. The average interpolated value (*fig. 9*) in the attrition cone is 0.85 mm. Al (Table XIV)

showing a 4 percent increase in radiopacity relative to the normal coronal dentine whose average interpolated value is 0.82 mm. Al. In the large cone of secondary dentine, average interpolated values of 0.82, 0.82, and 0.80 mm. Al are found which do not differ significantly from those of the normal dentine. However, the root dentine, average interpolated value of 0.77 mm. Al, is 5 percent less radiopaque than the crown.

Tooth No. 23: A lower right canine from a 41 year old male (same individual as Tooth No. 19), shows (*fig. 10*) a clearly defined, elongated, slender cone of transparent secondary dentine set like an arrow head with its base at the pulp chamber and its tip near the apex of a relatively small attrition cone. A roentgenogram of the 0.85 mm. thick section is studied. The average interpolated values (*fig. 9*) in the attrition cone is 0.85 mm. Al, a value showing an increased radiopacity of 5 percent relative to the normal coronal dentine, whose average interpolated values is 0.81 mm. Al (Table XV). The secondary dentine is 4 percent less radiopaque than the normal dentine. The root dentine is 2½ percent less radiopaque than the crown.

Tooth No. 54: A lower right incisor from a 47 year old female (same individual as Tooth No. 51), shows (*fig. 10*) a moderate attrition of the tip with a prominent cone of transparent secondary dentine with its base at the tip of the pulp chamber and its tip at the apex of the tiny attrition cone. The interpolated values (*fig. 9*) are obtained (Table XVI) from the roentgenogram of an 0.84 mm. thick section. The average interpolated value in the attrition cone is 0.86 mm. Al, a value 6 percent greater than the average interpolated values of 0.81 mm. Al found in the normal coronal dentine. The secondary dentine also shows a radiopacity 4 percent greater than the normal dentine. The root dentine, average interpolated value of 0.78 mm. Al is 4 percent less radiopaque than the crown.

SUMMARY

In summary, it is noted that in these 4 teeth showing a prominent cone of transparent secondary dentine and a tiny attrition cone: (1) the average interpolated values for the attrition cone are 4, 5, and 6 percent greater than normal coronal dentine; (2) the secondary dentine differs from the normal by $-2\frac{1}{2}$, -1, -4, and $+4$ percent; and (3) the root dentine is less radiopaque than the crown by 0, 5, $2\frac{1}{2}$, and 4 percent.

For the purpose of this discussion the teeth showing attrition have been grouped into three classes on the basis of similarities in roentgenographic appearance and absorption measurements, supplemented by their appearance when observed grossly using transmitted light. When these groups are compared with a normal young tooth, the interesting conjecture is made that these classes represent stages in the change which takes place in dentine exposed by attrition. Thus we think that slight attrition of the dentine might produce a prominent attrition cone and a laying down of a small amount of secondary dentine in the tip of the pulp chamber; that advancing attrition and

continued exposure of the tubules is accompanied by an increased amount of secondary dentine in the pulp chamber, giving rise to the "hour glass" effect; that advancing attrition removes more and more of that part of the tooth which includes the attrition cone leaving only the prominent cone of transparent secondary dentine. Further credence is given to the aptness of this classification by considering the summary of average interpolated values given in Table XVII.

TABLE XVII

Summary of interpolated values

CLASS	TOOTH NUMBER	AGE	AVERAGE INTERPOLATED VALUES						
			Normal dentine	Attrition cone	Increase	Transparent secondary dentine	Difference from normal	Root dentine	Difference from coronal
					per cent		*per cent*		*per cent*
Normal	39	15	0.81					0.76	−6
Prominent attrition cone	53	47	0.78	0.86	10	0.80	+2½	0.79	+1
	49	47	0.79	0.83	5	0.79	0	0.78	−1
	52	47	0.79	0.85	7½			0.79	0
	55	47	0.76	0.82	7			0.75	−1
Equal sized attrition and secondary dentine cones	21	41	0.80	0.83	4	0.75	−6	0.77	−4
	7	41	0.77	0.83	7½	0.79	+2½		
	20	41	0.80	0.83	4	0.76	−5	0.75	−6
	22	41	0.80	0.81	1	0.76	−5	0.78	−2½
	26	41	0.80	0.82	2	0.73	−9	0.75	−6
Prominent secondary dentine cone	51	47	0.76			0.74	−2½	0.76	0
	19	41	0.82	0.85	4	0.81	−1	0.77	−5
	23	41	0.81	0.85	5	0.78	−4	0.79	−2½
	54	47	0.81	0.86	6	0.84	+4	0.78	−4

It is at least a coincidence that the 4 teeth showing a prominent attrition cone have the greatest percent increase in radiopacity in the attrition cone, while the 5 teeth with the *hour glass* and the 4 teeth with the prominent secondary dentine cone have only moderate increases in radiopacity of the attrition cone. The coincidence is increased by noting 1) that the group of teeth with prominent attrition cones have secondary dentine about as radiopaque as the normal,

2) that the *hour glass* type show markedly less radiopacity in the secondary dentine, while 3) those teeth having prominent secondary dentine cones show small differences from normal dentine. Furthermore, it is seen that in the normal young tooth # 39, the root dentine is unquestionably less radiopaque than the crown; this is also the case for the *hour glass* type and those showing a prominent secondary dentine cone whereas, in teeth showing slight attrition and prominent attrition cones, the root dentine does not differ appreciably from the crown.

Attrition cones are more radiopaque than the normal coronal dentine in the same tooth, attributable only to an increase in the calcific material in the changed area. Because the attrition cone penetrates too deeply into the dentine to be ascribed to an effect of the packing forces of mastication, the changed appearance may be caused by a chemical action of the saliva, a physiological action in the dentine or both. However, the increased radiopacity cannot be explained as an effect of soft tissue change or necrosis and it is highly probable that the calcific deposition has occurred in the lumen of the tubules or the tubule processes into the matrix of the dentine. The exact histological location of the additional calcific material cannot be found by the densitometric method since by it we measure only gross changes involving a comparatively large area.

All of the teeth showing prominent attrition cones are from the same individual. All of the teeth showing the *hour glass* effect are from the same individual. Of the 4 teeth showing prominent secondary dentine cones, 2 are from one individual, 2 from another. It is impossible to rule out the possibility that the differences in roentgen-ray absorption found between these groups may have been closely dependent on the state of health of the individuals concerned.

However, the grouping of the abraded teeth in the discussion is shown to be an apt division since the teeth with prominent attrition cones all have markedly increased radiopacity of the attrition cones, the teeth with the *hour glass* effect all have secondary dentine with marked less roentgen-ray opacity than normal crown dentine, while the teeth with the prominent secondary dentine cone show little or no difference between radiopacity of the secondary dentine and that of normal coronal dentine. These findings do not answer the question

of what happens to dentine exposed by attrition. They will serve their purpose if they direct attention to the possibilities offered by this method of study.

CONCLUSION

(1) A young tooth shows constancy of roentgen-ray absorption values throughout the crown.

(2) In certain teeth showing slight attrition, the attrition cone in the dentine between the abraded incisal tip and the pulp chamber shows a marked increase in roentgen-ray absorption compared to the normal coronal dentine in the same tooth.

(3) In certain teeth showing slight attrition, where there are nearly equal sized attrition and secondary dentine cones, the secondary dentine shows markedly less radiopacity than the normal crown dentine, whereas the attrition cone shows only slightly increased absorption of roentgen-rays.

(4) In certain teeth showing moderate attrition with a prominent secondary dentine cone, the secondary dentine shows little difference in radiopacity from that of the normal and the attrition cone, where measurable, moderately increased radiopacity.

(5) In the 14 teeth studied, root dentine generally is less radiopaque than crown dentine in the same tooth.

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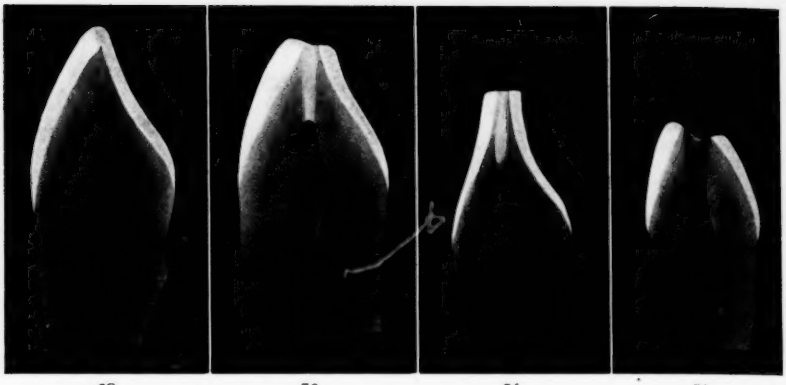
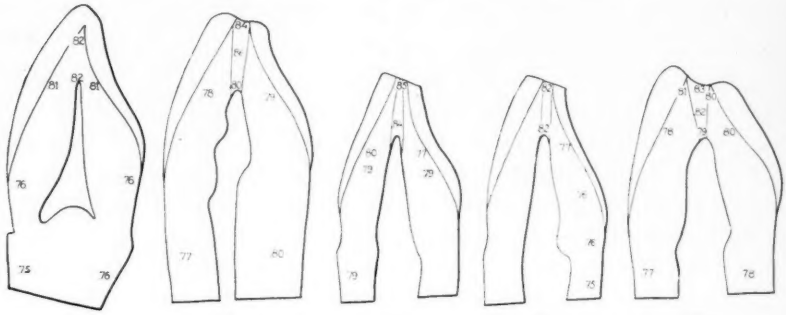


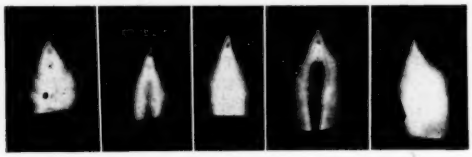
39 53 21 51

Fig. 4. Teeth Nos. 39, 53, 21, 51. Half-tone reproductions of x-ray images. No. 39 is young tooth with intact enamel and homogeneous x-ray absorption throughout dentine. No. 53 shows relatively large radiopaque attrition cone and tiny dot of transparent secondary dentine. No. 21 shows almost equal sized radiopaque attrition and radiolucent secondary dentine cones giving *hour-glass* appearance. No. 51 shows tiny attrition cone and large secondary dentine cone of almost normal radiopacity.



39 53 52 55 49

Fig. 5. Outline drawings of teeth Nos. 39, 53, 52, 55, and 49 with average interpolated values inserted in areas measured (compare Tables III, IV, V, VI, VII). No. 39 shows uniform values about 0.82 mm. Al in crown and 0.76 mm. Al in root. The other teeth show attrition cones of greater radiopacity (by 5 to 10 percent) than normal dentine in same tooth.



49 55 52 53 39

Fig. 6. Contact prints of teeth Nos. 49, 55, 52, 53, and 39. No. 39 is normal young tooth with intact enamel and homogeneous appearing dentine. The other teeth are classed as having prominent attrition cones and tiny transparent secondary dentine cones. Transparent secondary dentine cones show up as black triangles in contact prints.

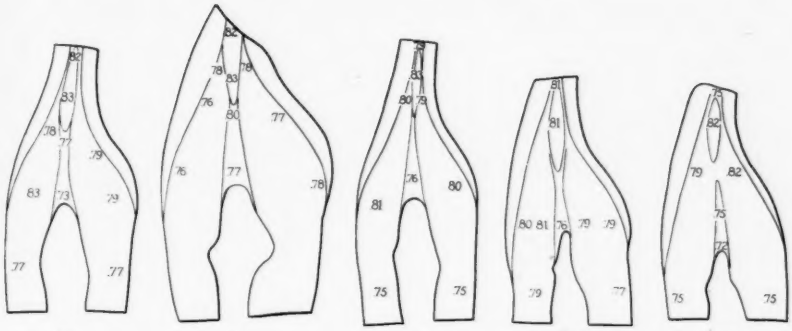


Fig. 7. Outline drawings of teeth Nos. 21, 7, 20, 22, and 26 with average interpolated values inserted at approximately the areas measured (compare Tables VIII, IX, X, XI, XII). In these teeth with nearly equal sized attrition and transparent secondary dentine cones giving an *hour-glass* appearance, attrition cones are of greater radiopacity (by 1 to 7½ percent), transparent secondary dentine cones are of lesser radiopacity (by 2½ to 9 percent) than normal coronal dentine in same tooth.

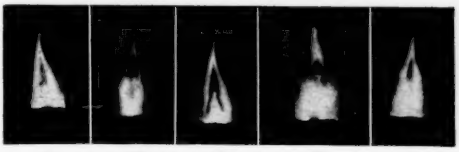


Fig. 8

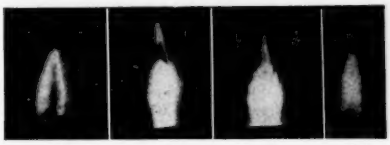


Fig. 10

Fig. 8. Contact prints of teeth Nos. 26, 22, 20, 7, and 21. Equal sized attrition and transparent secondary dentine cones are shown. In certain teeth, transparent secondary dentine cone has an arrow head appearance.

Fig. 10. Contact prints of teeth Nos. 54, 23, 19, 51 showing relatively large transparent secondary dentine cones and small attrition cones.

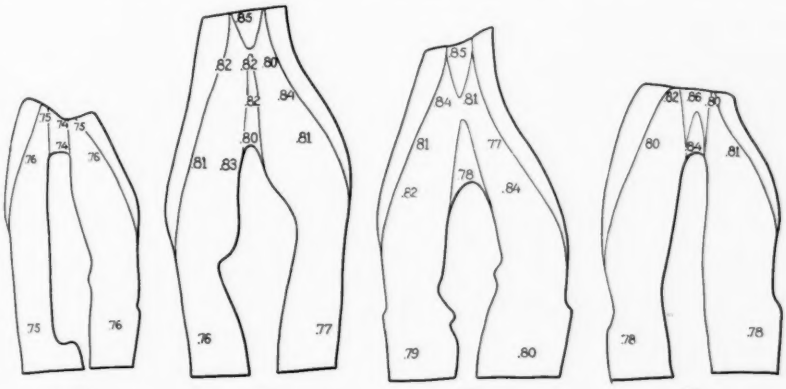


Fig. 9. Outline drawings of teeth Nos. 51, 19, 23, and 54 giving average interpolated values at approximately areas measured. Prominent transparent secondary dentine cones show lesser radiopacity (by 1 to 4 percent), small attrition cones show greater radiopacity (by 4 to 6 percent) than normal coronal dentine in same tooth.